A STUDY ON PLATELET VOLUME INDICES IN

ACUTE CORONARY SYNDROME

Dissertation submitted to

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In partial fulfilment of regulations

For award of the degree of

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BONAFIDE CERTIFICATE

This is to certify that dissertation named "***A STUDY OF PLATELET VOLUME INDICES IN ACUTE CORONARY SYNDROME"*** is a bonafide work performed by **Dr.MANIKANDAN.S,** post graduate student, Department of Internal Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision in fulfilment of regulations of the Tamil Nadu Dr. M.G.R Medical University for the award of M.D. Degree Branch I (General Medicine) during the academic period from 2013 to 2016.

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DECLARATION

I solemnly declare that the dissertation entitled *"**A STUDY ON PLATELET VOLUME INDICES IN ACUTE CORONARY SYNDROME**"* is done by me at Kilpauk Medical College, Chennai from January 2015 to August 2015 under the guidance and supervision of Prof. K.V.RAJALAKSHMI, M.D., to be submitted to The Tamilnadu Dr M.G.R Medical University towards the partial fulfilment of requirements for the award of M.D DEGREE IN GENERAL MEDICINE BRANCH-I.

Place: Chennai

Date: (Dr. S. MANIKANDAN)

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ABBREVIATIONS

CAD - Coronary artery disease

AP - Angina pectoris

ACS - Acute coronary syndrome

AMI - Acute myocardial infarction

UA - Unstable angina

STEMI - ST elevation MI

NSTEMI - Non ST elevation MI

HT - Systemic Hypertension

DM - Diabetes mellitus

WBC - White blood cell

TC - Total count

P - Polymorphs

L - Lymphocytes

E - Eosinophils

TB - Total bilirubin

DB - Direct bilirubin

SGOT - Aspartate transferase

SGPT - Alanine transferase

MPV - Mean platelet volume

PDW - Platelet distribution width

P-LCR - Platelet – Large cell ratio

TC - Total cholesterol

TGL - Triglycerides

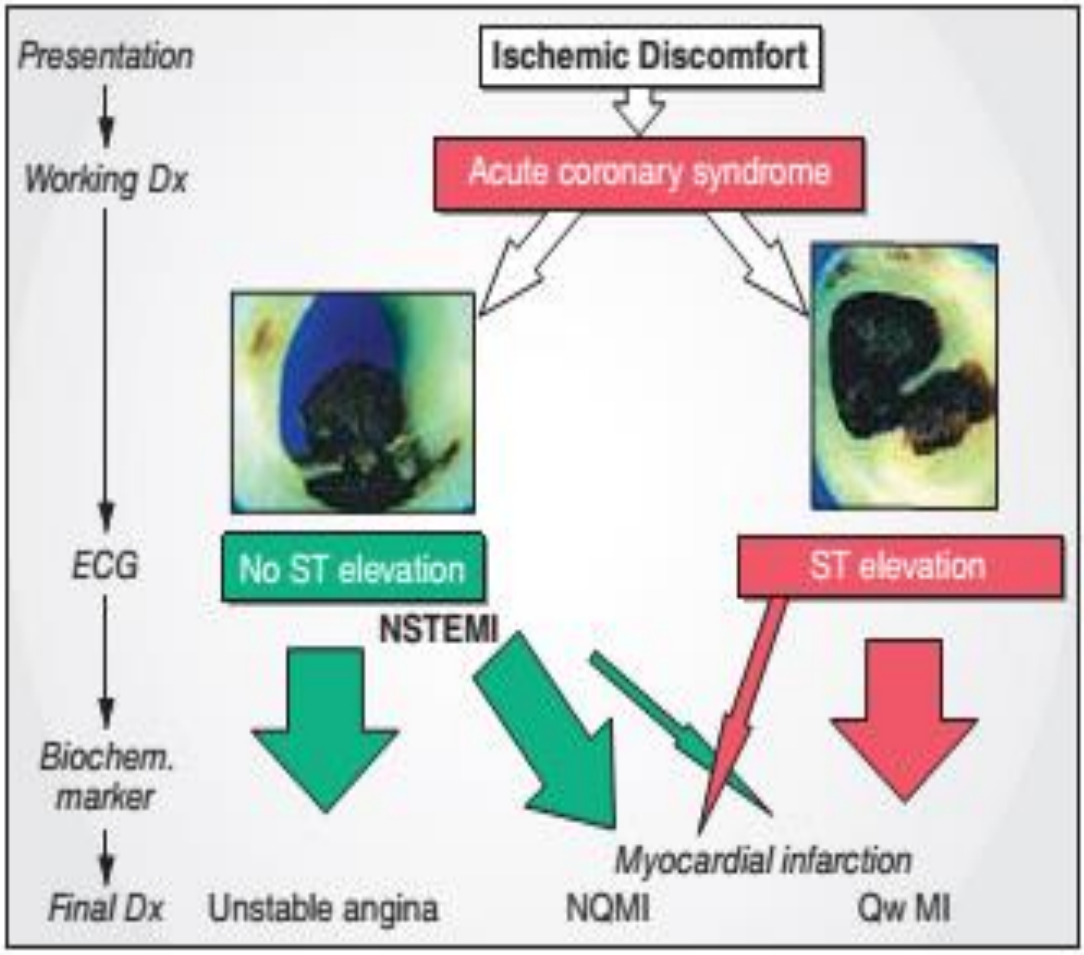
HDL - High density cholesterol

CONTENTS

INTRODUCTION

Acute coronary syndrome is one of the leading cause of death and disability even though the development of recent advances in its medical science.

Coronary heart disease may present as silent myocardial infarction, stable angina (AP), unstable angina (UA), myocardial infarction (STEMI and NSTEMI), sometimes heart failure and rarely sudden cardiac death.



Most important step in the management of acute coronary syndrome is to identify the risk factors, as identification of these risk factors and avoidance of these risk factors helps in the prevention of the development of myocardial ischemia.

Followed by the ruptured plaque, platelets play a vital role in the development of thrombus and further progression to myocardial infarction. Hence, by using cyclooxygenase II inhibitor and glycoprotein IIb/IIIa inhibitor (ticlopidine), inhibition of platelet action is used in the management of ACS.

By altering one of the parameters i.e., density, size or activity, this leads to triggering of acute coronary syndrome and its spread. Smaller platelets are less adhesive, less active and tend to aggregate less easily than larger ones. The prothrombotic tendency of atherosclerotic plaque in acute coronary syndrome is found to be increased in those who have increase in platelet volume and this leads to increased incidence of thrombus formation in such patients.

Our aim was to investigate significance of platelet volume indices in acute coronary syndrome in this study.

AIMS OF THE STUDY

- To investigate the importance and role of platelet volume indices in acute coronary syndrome.

- To compare platelet volume indices in ACS (Acute myocardial infarction, unstable angina), stable angina pectoris and healthy controls.

REVIEW

OF

LITERATURE

REVIEW OF LITERATURE

CORONARY ARTERY DISEASE is one of the leading causes of death and disability in any part of world. The better known risk factors are

1) Age
2) Family history of prior CAD
3) Cigarette smoking
4) Dyslipidemia
5) High body mass index
6) Physical inactivity
7) Hypertension
8) Diabetes mellitus which is accepted to be a coronary heart disease equivalent.

Apart from these, novel risk factors includes

a) Lipoprotein (a)
b) Endothelial dysfunction
c) Homocysteine
d) C Reactive protein
e) IL-6 and membrane bound IL-6 receptors

f) Increased activity of leukocyte enzyme myeloperoxidase

g) Metabolic syndrome is considered as a new risk factor of coronary heart disease.

All these risk factor are found to be associated only with minority of ACS cases. Hence, it is important to detect associated risk factor to predict the development of ACS on an individual basis.

ACUTE MYOCARDIAL INFARCTION[1]

Aspects of Diagnosis of Myocardial Infarction by different Techniques

TECHNIQUE	FEATURES
Pathology	Myocardial cell death
Biochemistry	Markers of myocardial cell death recovered from blood samples
Electrocardiography	Evidence of myocardial ischemia (ST and T wave abnormalities); evidence of loss of electrically functioning cardiac tissue (loss of R wave)
Imaging	Cardiac wall motion abnormalities ;Reduction or loss of tissue perfusion

REVISED DEFINITION OF MYOCARDIAL INFARCTION[1]

One of the following criteria to be fulfilled for diagnosing acute, evolving, or recent MI:

1) Increase or decrease in cardiac biomarkers during myocardial injury along with at least one of the following:
    a. Clinical features of ischemia
    b. Presence of pathologic Q waves in the ECG

c. Presence of new presumed significant ST-segment changes or new LBBB

d. Demonstration of new loss of viable tissue in myocardium in cardiac imaging or echocardiography showing new regional wall motion dysfunction

e. Demonstration of intracoronary thrombus by autopsy or angiography.

2) Tissue biopsy shows myocardial cell death.

HEALING OR HEALED MYOCARDIAL INFARCTION[1]

For diagnosis of healing or healed myocardial infarction, anyone of the following criteria to be fulfilled:

1) Serial ECG of patients shows new pathologic Q waves. The patient may or may not remember previous symptoms. Biochemical markers of myocardial necrosis may have normalized, depending on the length of time that has passed since the infarction developed.

2) Imaging shows loss of viable myocardium which is thinned and not able to contract, without nonischemic cause.

3) Myocardial tissue biopsy shows healed or healing infarction .

PATHOLOGY OF AMI[2:]

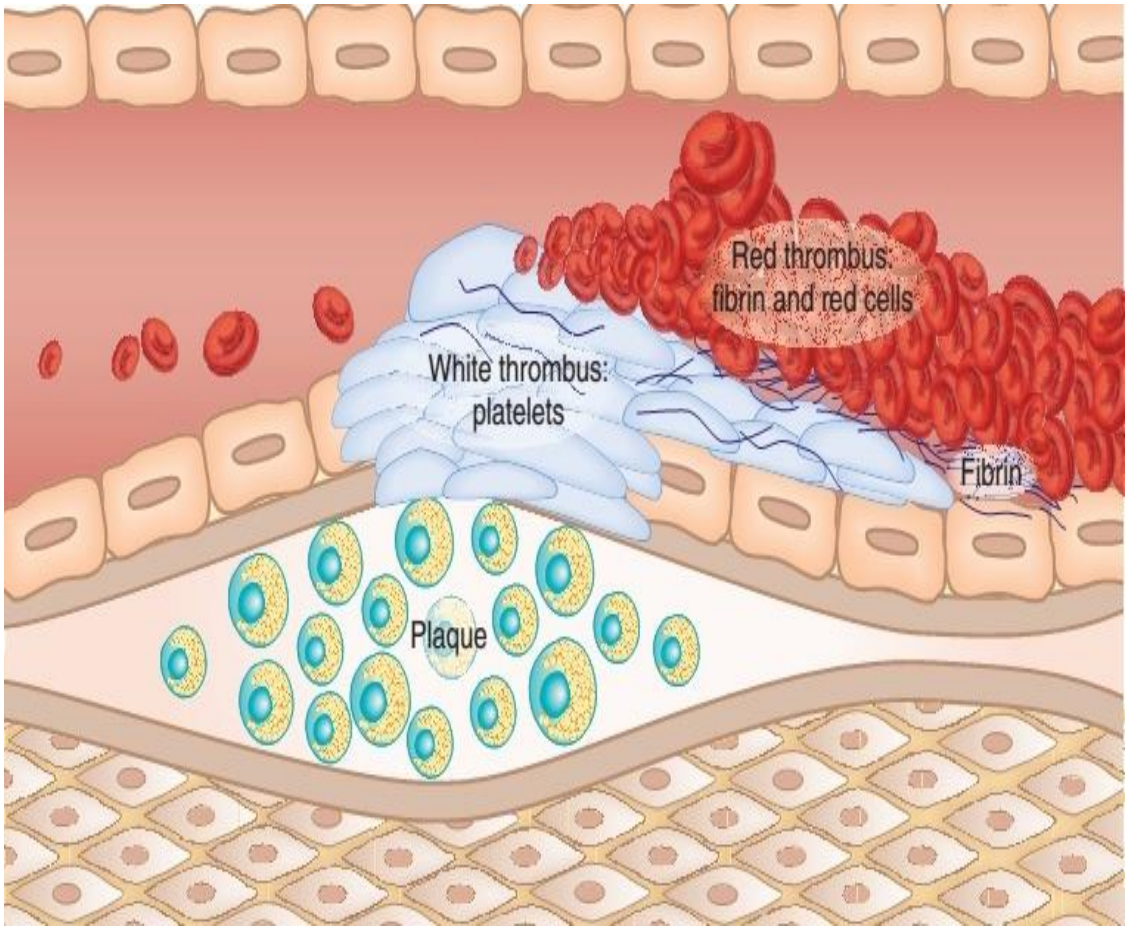
In a coronary artery already affected by atherosclerosis thrombotic occlusion, leads to sudden decrease in coronary blood flow which results in formation of STEMI .

STEMI does not occur in a gradually developing, coronary artery occlusion because over a period of time there is development of rich collateral blood flow.

At a site of vascular injury when there is rapid development of coronary artery thrombus, STEMI occurs. This injury is produced or favoured by risk factors such as alcohol intake, dyslipidemia, smoking and hypertension[2].

Formation of thrombus occurs in two settings: 1) release of content of an atherosclerotic plaque once its surface gets disrupted 2) local or systemic conditions which favors thrombogenesis exist. In the presence of above conditions, mural thrombus is formed at this site of plaque rupture, and finally results in occlusion of coronary artery. Coronary plaques which have rich lipid core and a thin fibrous cap are found to be more prone to disruption by histological studies [2].

At the site of plaque rupture, initially platelet monolayer is formed which leads to release of various agonists promoting activation of platelet like collagen, adenosine diphosphate, epinephrine, 5-hydroxytryptamine. After activation of platelets by these agonists which leads to release of potent vasoconstrictor- Thromboxane A_2, which causes further activation of platelet and potential resistance to fibrinolysis is developed[2].



Production of potent vasoconstrictor Thromboxane A_2 and activated platelet cause change in conformational in glycoprotein IIb/IIIa receptor and then it is converted to its functional state, which has high affinity for soluble adhesive proteins-fibrinogen. Multivalent nature of fibrinogen helps in simultaneously binding with two different platelets at the same time, promoting aggregation of platelets and their cross-linking[2].

At the site of the plaque rupture, damaged endothelial cells release tissue factor. On exposure of tissue factor, the coagulation cascade is activated. Activation of extrinsic pathway of coagulation cascade leads to release of Factors VII and X, finally leading to the conversion of prothrombin to thrombin, which eventually results in formation of fibrinogen to fibrin. The formed thrombus occludes the involved coronary artery.[2]

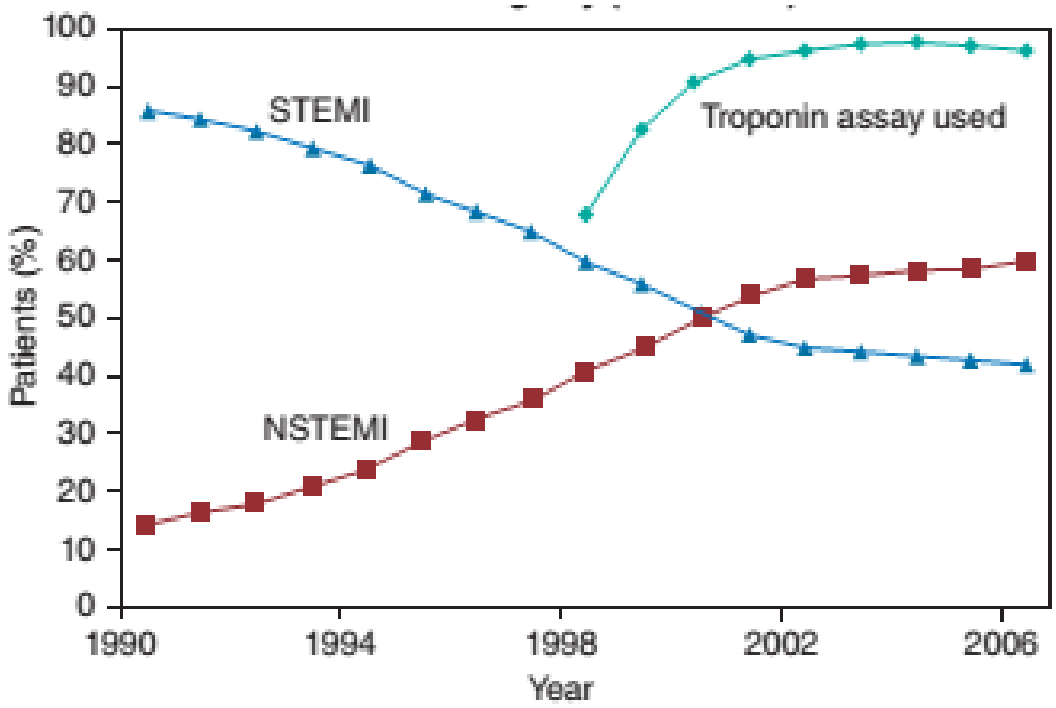
DEFINITION[1]

STABLE angina pectoris usually manifests as a deep, ill defined localized chest discomfort radiating to left arm (sometimes presented as pain), which is aggravated by physical activity or emotional stress, and relieved by rest or nitrates .

UNSTABLE angina manifests as angina pectoris (or similar type of ischemic discomfort) which is associated with anyone of following three features:

1) Severe chest pain occurring at rest or minimal exertion, lasting for >20 minutes and prolonged more unless interrupted by the ingestion of a sublingual nitrate.
2) New onset (within 1 month) severe frank chest pain.
3) Follows a crescendo pattern I) that is pain that is severe enough to awaken the patient suddenly from sleep ii) that is more severe in nature , prolonged duration , or frequent than before.

Approximately two thirds of patients with unstable angina are diagnosed as NSTEMI on the basis of elevated serum cardiac markers, like cardiac-specific troponin T or I and creatine kinase isoenzyme CK–MB, as these are evidence of myocardial necrosis. NSTE-ACS exhibit release of troponin, and as troponin is becoming progressively more sensitive. Hence many patients are diagnosed as NSTEMI with a reciprocal reduction in the fraction with unstable angina.



PATHOLOGY OF UNSTABLE ANGINA/NSTEMI[2]

UA/NSTEMI is most commonly caused by an increase in myocardial oxygen demand and /or decrease in oxygen supply which is superadded on a lesion, usually an atherothrombotic coronary plaque, that causes coronary arterial occlusion.

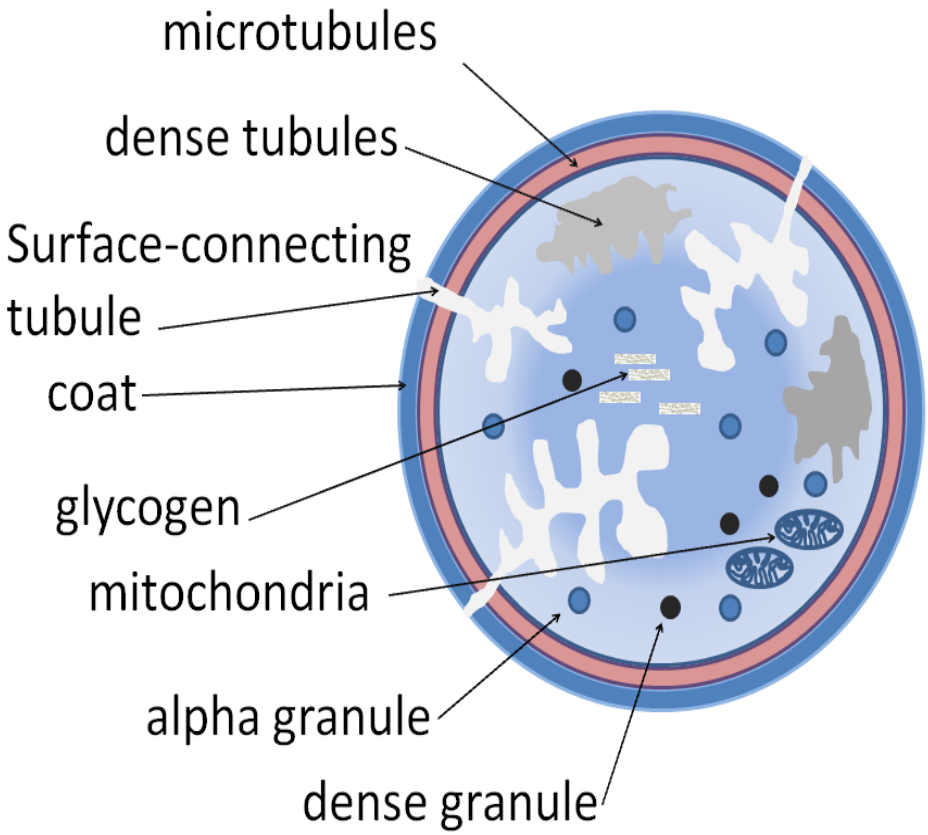
Four pathological processes that may contribute to the formation of UA/NSTEMI has been determined[2]:

1) Rupture or erosion of atherosclerotic plaque which results in formation of nonocclusive mural thrombus, is the most common pathology in such patients, NSTEMI may occur with embolization of atherosclerotic debris and/or platelet aggregates downstream.
2) Intermittent dynamic obstruction e.g., coronary artery vasospasm seen in Prinzmetal's variant angina (PVA)
3) Mechanical obstruction e.g., Restenosis following PCI percutaneous coronary intervention or coronary atherosclerosis which is rapidly progressive .
4) Unstable Angina which is due to increased myocardial stress (oxygen supply mismatch) e.g., tachycardia, anemia.

Single factor or combination of factor is involved.[2]

PLATELETS[3]

Platelets are formed from precursors megakaryocytes in the bone marrow . They are disc-shaped, enucleate cell fragments, which are released from the bone marrow into the blood circulation. Following a vascular injury platelet help in the formation of hemostatic plug that initially seals vascular defect. They also provide a surface for recruiting and concentrating coagulation factors which further enhances the coagulation cascade. Thus platelets play a critical role in normal haemostasis.



The function of platelets depends upon

► Several glycoprotein receptors,

►a contractile cytoskeleton, and

►two types of cytoplasmic granules: a) α-*Granules*

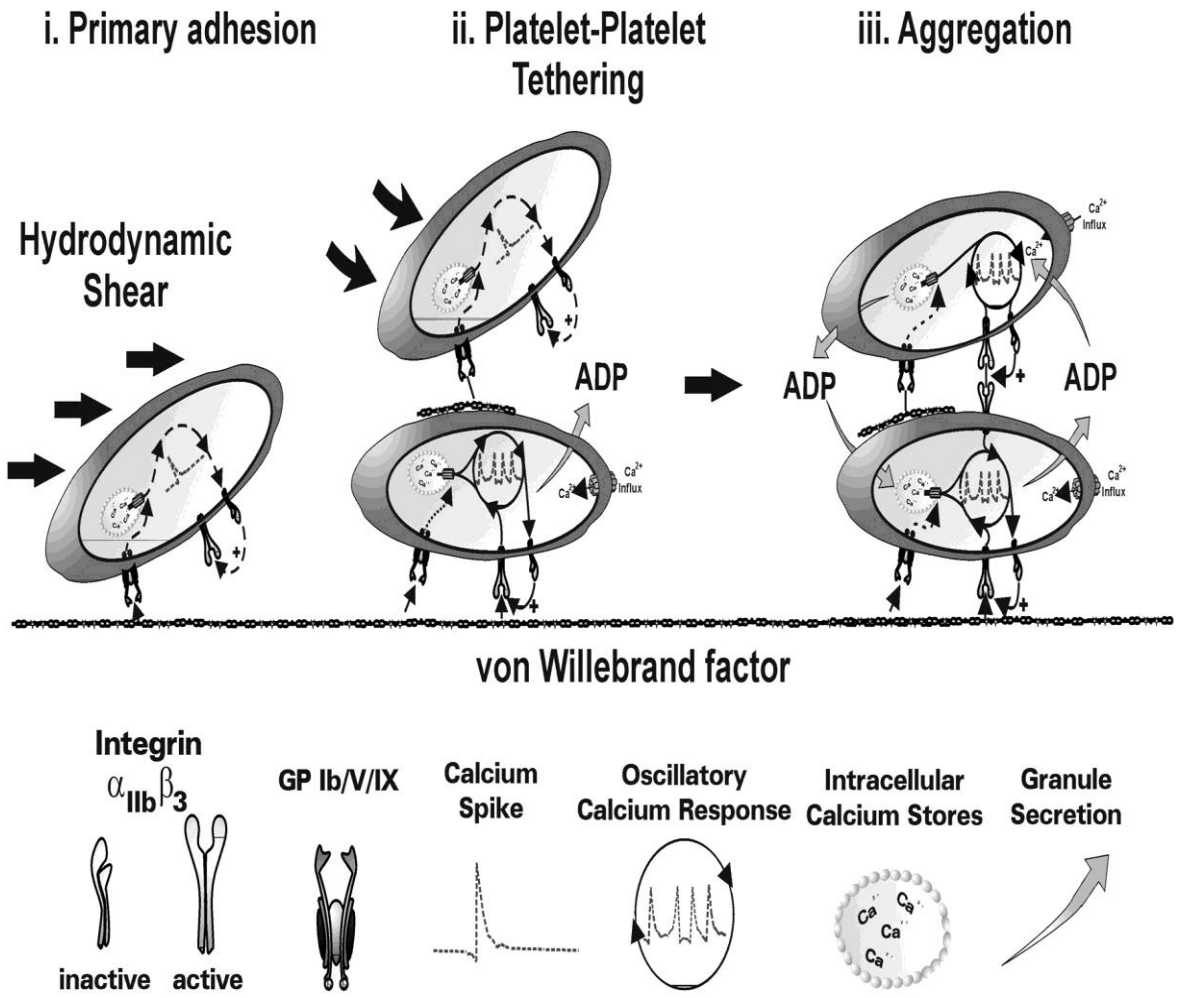
b) Dense (or δ) granule

After vascular injury, platelets come in contact with extra cellular matrix contents such as collagen and the adhesive glycoprotein vWF. On contact with these proteins, platelets undergo the following physiological changes like:

1) Platelet adhesion and shape change,

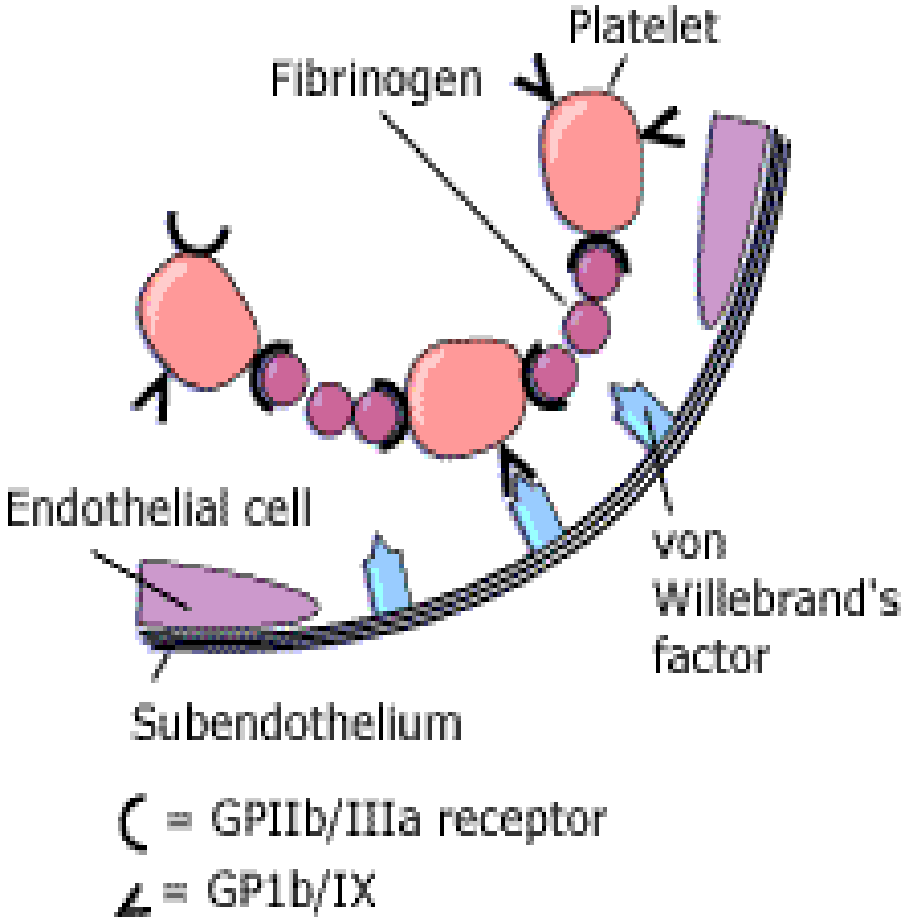
2) Secretion (release reaction) and

3) Aggregation



Adhesion of platelets with extra cellular matrix is mediated via interactions with vWF. vWF acts as a bridging connection between glycoprotein Ib [GpIb] (platelet surface receptors) and exposed collagen. Platelets can adhere to various components of extracellular matrix like fibronectin but the vWF-GpIb associations are essentially necessary because only this association can withstand the high shear forces of

flowing. Thus genetic deficiencies of vWF or its receptor result in bleeding disorders depicting importance of these interactions. Deficiency of this receptor defect is referred to as Bernard-Soulier syndrome.

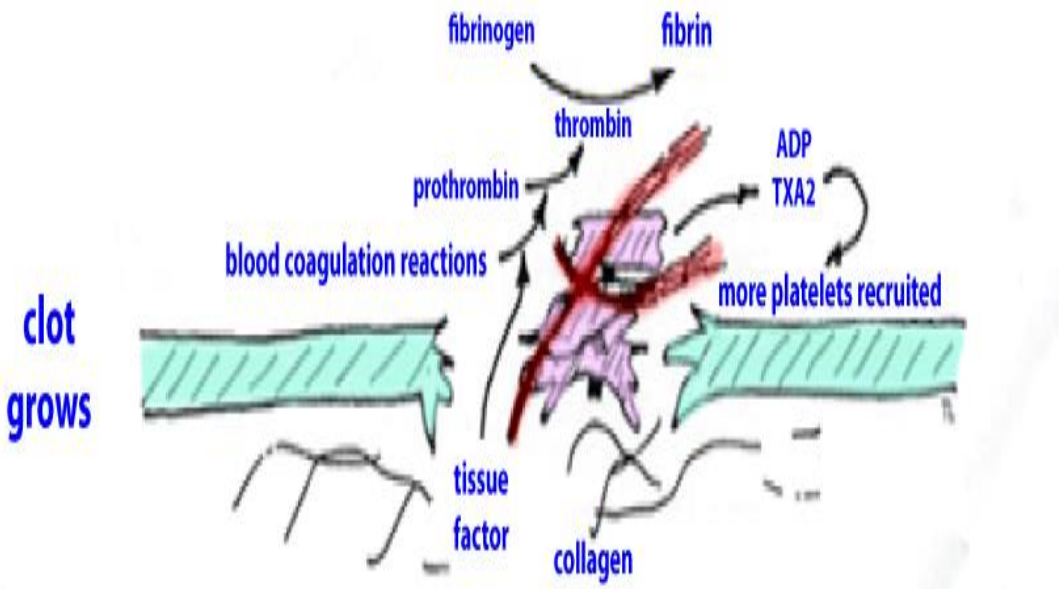


Soon after adhesion secretion takes place . Secretion is also called as release reaction. There is release of both alpha and dense granule. Intracellular protein phosphorylation cascade is stimulated by interaction of various agonists with platelet surface receptors and followed by degranulation. Calcium and ADP are contents of dense-bodies and there

role in coagulation is of utmost important, because calcium acts as activator in coagulation cascade, and ADP promotes further aggregation. ADP helps in further amplification of coagulation cascade because ADP promotes further ADP release.

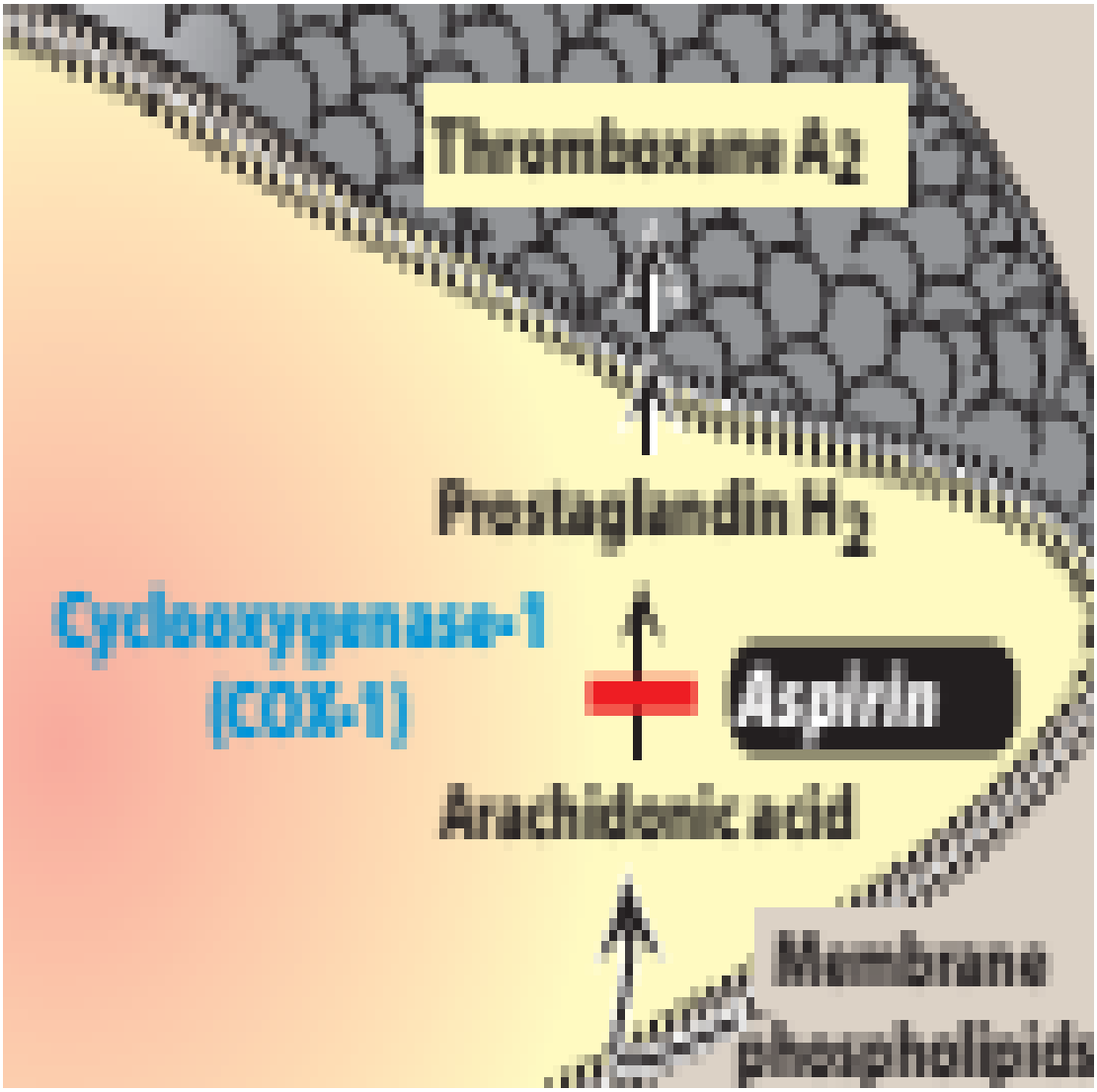
Ultimately, activation of platelets results in the exposure of negatively charged phospholipids (mainly phosphatidylserine) on their outer surfaces which in turn interact with calcium and plays as nucleation sites for the assembly of coagulation factors complexes [3].

Platelet aggregation is followed by adhesion and release of granule. Like ADP that amplifies platelet aggregation, Thromboxane A_2 another platelet-derived vasoconstrictor which also amplifies platelet aggregation. Thus the three processes adhesion, secretion and aggregation results in the formation of the primary hemostatic plug. This initial cascade of aggregation is reversible, but with generation of thrombin, simultaneous activation of the coagulation cascade results in stabilization of the platelet plug . Thrombin causes stabilisation of platelet plug in two ways:



(1) Thrombin causes further platelet aggregation and activation by binding with protease-activated receptor on the platelet membrane and also by its interaction with ADP and TxA_2 . The cytoskeleton of platelet plays a major role in activation of platelet by forming an irreversibly fused mass of platelets, which results in the definitive secondary haemostatic plug[3].

(2) Thrombin mediates the conversion of fibrinogen to fibrin, which functionally cements the platelets in place.



The interaction between platelets and endothelium has a profound impact on clot formation. Endothelial cell-derived factor- Prostacyclin (PGI2) is a potent vasodilator and inhibits platelet aggregation, whereas TxA2 is a potent vasoconstrictor and activates platelet aggregation. Effects of PGI_2 and TxA_2 should be well balanced effectively as they modulate platelet and vascular wall function.

Eventually by the effect of the above endothelial cell-derived factor, platelet aggregation is prevented and hemostatic plug formation takes place. Irreversible cyclooxygenase inhibitor – aspirin has the ability to permanently block platelet TxA_2 synthesis and hence it is clinically utilised in persons at risk for coronary thrombosis. Aspirin inhibits endothelial PGI_2 production, thereby prevents platelet plug formation but endothelial cells can overcome this blockage by resynthesizing cyclooxygenase enzyme.

MEAN PLATELET VOLUME

Platelets are heterogeneous regarding their size, density, and functional activity[19]. Alterations of these parameters results in pulling the trigger of acute coronary syndrome and its spread[20].

In ACS, thrombogenic phenomenon begins with the rupture of atherosclerotic plaque. The functions of circulating platelets are necessary for the thrombogenic phenomenon in ACS[21].

Test like in vitro aggregometry, has found significant difference in the function of large and small platelets. Compared to smaller platelets large platelets are more adhesive and aggregative. Large platelets contain higher levels of P-selectin and glycoprotein IIIa which are procoagulatory surface proteins. Thus in ACS, prothrombotic tendency of atherosclerotic plaque increases proportionality with increase in mean platelet volume and is also associated with increased risk of intracoronary thrombus formation in acute myocardial infarction cases[5].

Platelet volume has a major role in platelet function and activation. More secretary granules and mitochondria are present in larger platelets compared to small platelets thus large ones are more active.

Larger platelets are hyperactive leading to the formation and embolisation of intracoronary thrombus and thus large platelet promotes the emergence of acute coronary syndrome.

Thus Increased platelet volume measured after MI, has been suggested to be a risk factor for further ACS episodes.

Along with the other cardiac biomarkers, usefulness of MPV is such that it can to be used as a routine test for the risk stratification of ACS in patients admitted to the ICCU. Major advantage of PVI is that it is a simple and inexpensive laboratory measurement.

Due to alterations in the autonomic nervous system MPV has great diurnal and nocturnal variation as found out by studies.

A study also showed that there is a correlation between sympathetic nervous activity and the MPV in AMI patients as the adrenergic system exerts its effect on thrombopoiesis in bone marrow and peripheral platelet activation.

1) The effects of the adrenergic system on platelet activation take place in two ways in the peripheral circulation. $Alpha_2$-adrenoreceptor activation results in change of the shape of platelets and hence increases MPV.

2) Following exercise or following administration of adrenaline, larger, activated platelets which are sequestered in the spleen are released into the circulation leading to the increase in MPV following physical effort[46].

It has been shown that during admission in AMI patients high MPV can be used as an independent risk factor for risk stratification regarding impaired perfusion and associated death.

MEAN PLATELET VOLUME[45]

The mean platelet volume result is calculated by an automated analyser. MCV is a calculation of the mean size of individual red blood cells whereas MPV is a calculation of the mean size of platelets.

Normal Range7.5-11.5 femtolitre

If MPV is reduced

It indicates the platelets are smaller than usual size. They are referred to as Micro thrombocytes[45].

CAUSES:-

1. Aplastic Anemia-

An acquired disorder in which the new blood cells production is halted in the bone marrow.

2. Wiskott-Aldrich Syndrome-

An inherited immune deficiency disorder.

3. Thrombocytopenia-absent radii (TAR Syndrome) -

A rare inherited disorder in which there is absence of the radius bones in the forearms associated with low platelet count.

4. Storage Pool Disease

If MPV is increased

It indicates that platelets are larger than normal. They are referred to as Macro thrombocytes[45].

CAUSES:-

1. Idiopathic Thrombocytopenic Purpura-

A bleeding diathesis in which there is disorder of blood clotting mechanism.

2. Bernard-Soulier Disease -

An inherited disorder where the function of platelets are deficient in help to adhere to the walls of the blood vessels.

3. May-Hegglin Anomaly -

A rare, inherited disorder in which there are abnormally large platelets.

4 Greater risk of heart attacks and stroke-

As there are increased numbers of hyperaggregable large platelets there is increased risk of ischemic events.

MPV is found to be elevated in patients with acute coronary syndrome at the time of admission in coronary care unit , and it is being hypothesised that changes in the entire megakaryocytic-platelet-haemostatic axis precedes acute coronary events.

It s postulated that MPV increases before MI for three reasons[46]:

1) The life span of platelets is eight days approximately, and the increase in MPV is noted within the first 12 hr of admission

2) The increase in MPV persists beyond six weeks after discharge during which time the infarct would be largely healed.

3) Log normality of platelet volume is preserved.

An alpha1-adrenoreceptor antagonist Doxazosin, or an angiotensin II receptor blocker Losartan have been found to exert therapeutic effects on platelet size in vitro based on the observations of recent studies which have not yet been confirmed. In a study conducted with small number of 30 patients, revealed that platelet aggregation inhibitors have no effect on MPV values.

MATERIALS

AND

METHODS

MATERIALS AND METHODS

SETTING

This study is conducted in Cardiology department (CCU & OP) and Department of Medicine, Government Royapettah Hospital in collaboration with Department of Pathology and Biochemistry.

ETHICAL APPROVAL

Obtained.

STUDY DURATION

This study was conducted over a period of 7 months from January 2015 to August 2015.

STUDY POPULATION

Patients admitted with acute coronary syndromes in coronary care unit, stable angina pectoris patients followed in cardiology op and medical op and healthy controls in medical wards and medicine op in Government Royapettah Hospital.

SAMPLING

Patients are allotted by convenience sampling in each group.

TYPE OF STUDY

It is a hospital based cross sectional study.

INCLUSION CRITERIA

Patients presenting with chest pain in the casualty/Medical OPD are evaluated and divided according to clinical manifestations, ECG and echo into four groups.

1. Non cardiac chest pain with no evidence of IHD in history, physical examination, ECG and Echo constitute normal group (controls).
2. Patients with clinical manifestations, ECG and Echo suggestive of UA/NSTEMI.
3. Patients with clinical manifestations, ECG and Echo suggestive of STEMI.
4. Patients with clinical manifestations, ECG and ECHO suggestive of chronic stable angina

EXCLUSION CRITERIA

1. Patients with bleeding disorders, blood dyscrasias, preeclampsia, sepsis.
2. Patients with history of blood transfusion recently (within 6 weeks)
3. Patients with history of major operations ,trauma recently (within 6 weeks)
4. Patients who are receiving drugs which can cause thrombocytopenia
5. Patients with infections known to cause thrombocytopenia.

SAMPLE SIZE: The study will include a total of **100** patients.

- **25** patients with **non cardiac chest pain**
- **25** patients with **UA/NSTEMI**
- **25** patients with **STEMI**
- **25** patients with **chronic stable angina**

METHODS

Informed consent will be taken from all patients included in this study. Blood samples would be drawn at the time of admission before initiation of treatment. Estimation of platelet count, MPV, PDW and PLCR will be calculated in all patients included in this study(by Hemogram). Processing of blood samples would be done within 30 minutes of blood collection using an autoanalyser.

Clinical history will include

1. age
2. sex
3. history of precipitating factors like physical exercise
4. emotional stress
5. medical disorder
6. surgical disorder
7. past history of diabetes
8. hypertension
9. smoking
10. alcohol
11. previous episodes of chest pain.

Clinical examination will include vitals, general examination and systemic examination including detailed examination of CVS.

Investigations include

a. Complete hemogram (including MPV, PDW and PLCR) using an autoanalyser.
b. RBS, urea, creatinine, bilirubin
c. Serum lipid profile in the fasting state.
d. ECG
e. Echocardiography

On the basis of history, physical examination and investigations patients will be divided into to four groups – those with

STEMI ,
UNSTABLE ANGINA/NSTEMI ,
NON CARDIAC CHEST PAIN AND
CHRONIC STABLE ANGINA.

The platelet indices of the four groups will be compared.

EQUIPMENTS AND MATERIALS

Facilities for ECG are available in the casualty itself. Hemogram will be done free of cost in the pathology lab of Govt. Royapettah Hospital . Facilities for echocardiography are available in the cardiology department.

STATISTICAL ANALYSIS

The tests used are

a. ANOVA
b. Chi-square test and
c. t test

Data were collected and statistical significance was analysed.

STATISTICAL

REPORTS

RESULTS

SEX AND ACS

TABLE 1.1:

SEX	FREQUENCY	PERCENTAGE
MALE	40	80%
FEMALE	10	20%
TOTAL	50	100%

DIAGRAM 1:

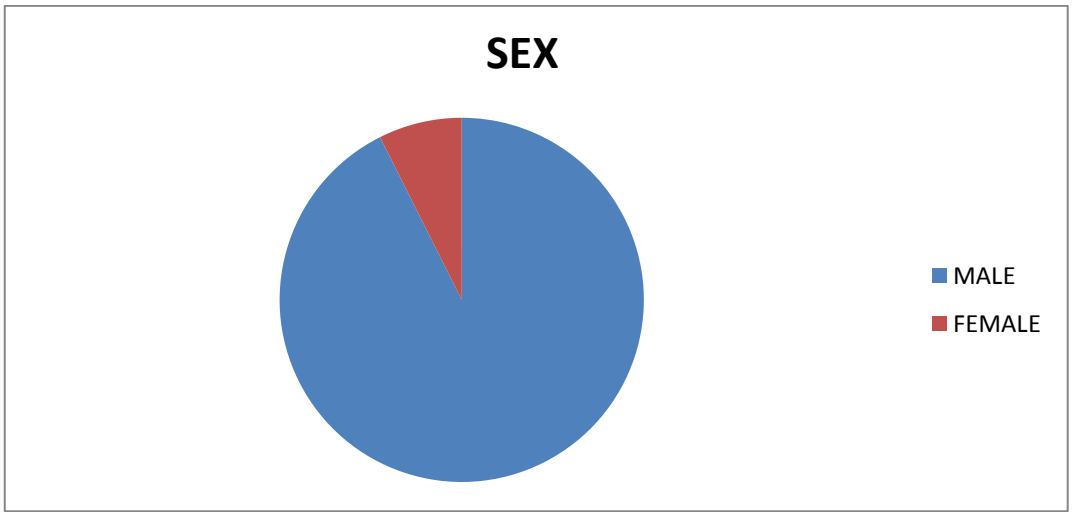


TABLE 1.2:

SEX	MEAN	N	STD DEVIATION	STD ERROR OF MEAN
MALE	9.570	40	.8715	.1378
FEMALE	9.960	10	.8113	.2566

TABLE 1.3:

MPV	Sig P Value
Between Groups	0.206 (NOT SIG)

AGE GROUP AND ACS

TABLE 2.1

AGE GROUP	FREQUENCY	PERCENTAGE
Below 40	10	20%
41-50	26	52%
51-60	11	22%
Above 60	3	6%
TOTAL	50	100%

DIAGRAM 2 :

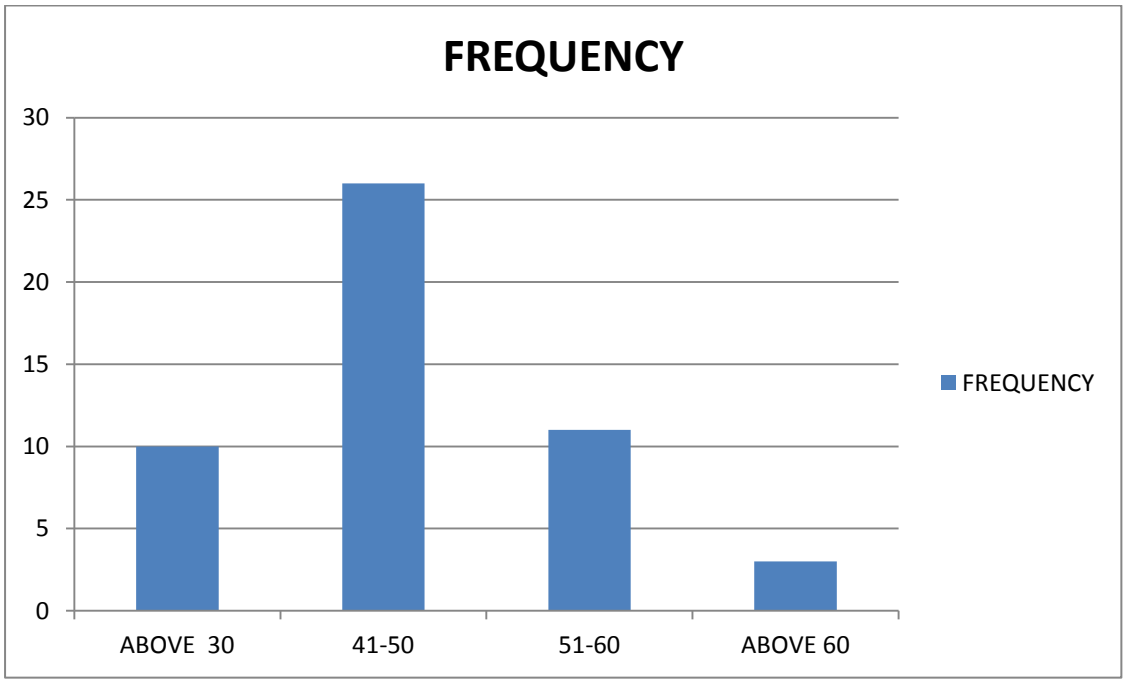


TABLE 2.2: AGE GROUP AND MPV

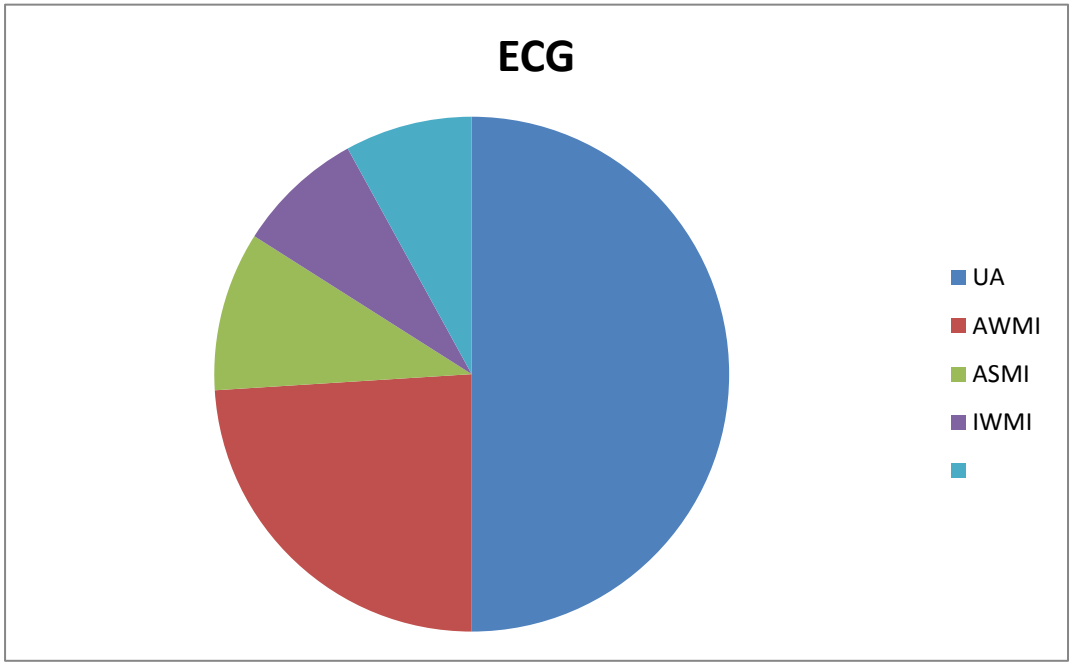
MPV	P Value
Between Groups	0.765 (NOT SIG)

TYPE OF ACS BASED ON ECG

TABLE 3:

MI	FREQUENCY	PERCENTAGE
AWMI	12	25%
ASMI	5	10%
IWMI	4	8%
IW + PWMI	4	8%
UA	25	50%
TOTAL	50	100%

DIAGRAM 3:



HYPERTENSION AND ACS

TABLE 4.1:

HYPERTENSION	FREQUENCY	PERCENTAGE
YES	31	62%
NO	19	38%

DIAGRAM 4:

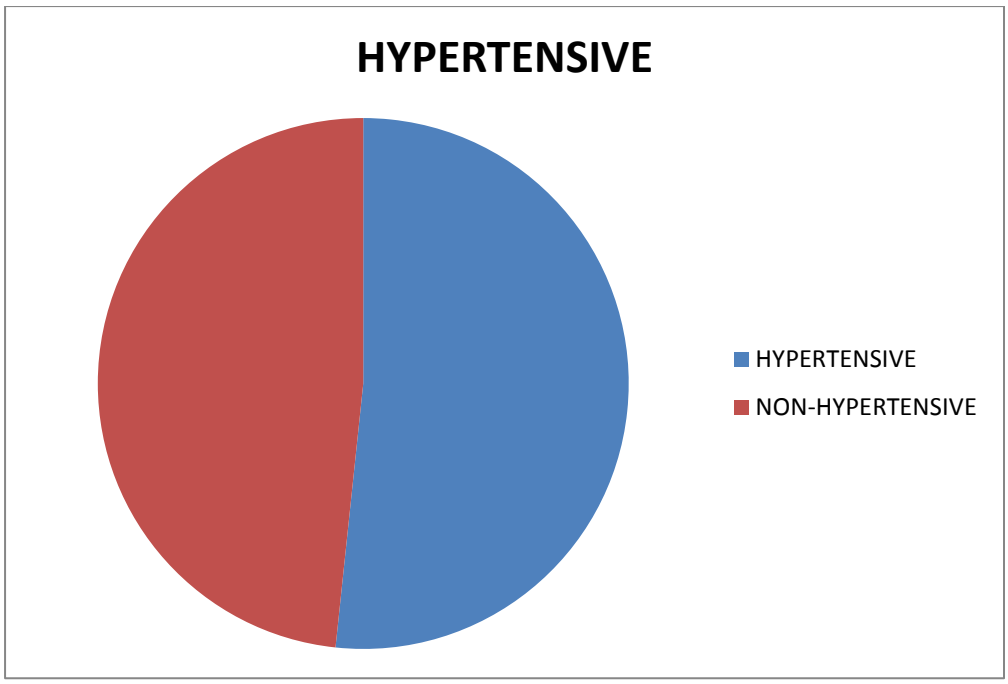


TABLE 4.2: HYPERTENSION AND MPV

MPV	Sig p value
Between Groups	0.346 (NOT SIG)

DIABETES AND ACS

TABLE 5.1:

DIABETIC	FREQUENCY	PERCENTAGE
YES	34	68%
NO	16	32%

DIAGRAM 5:

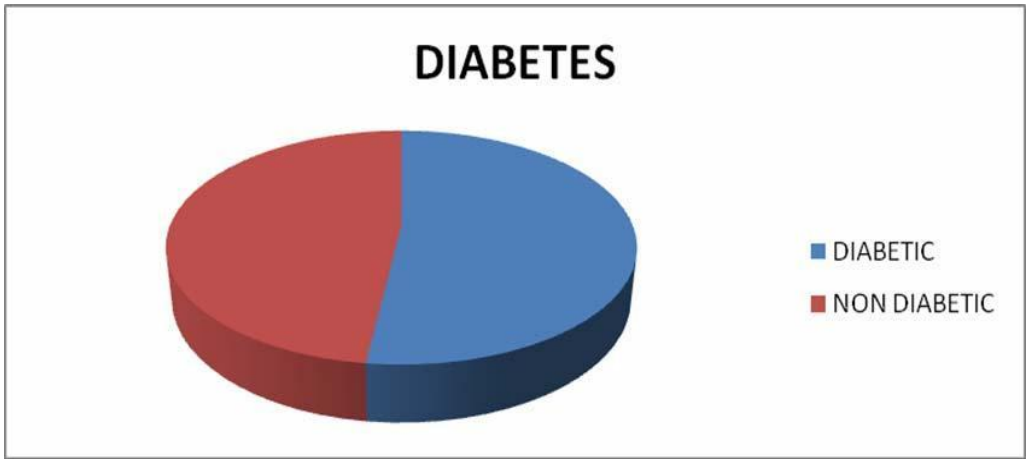


TABLE 5.2: DIABETES AND MPV

MPV	Sig P value
Between Groups	0.959 (NOT SIG)

PRIOR CAD AND ACS

TABLE 6.1:

PRIOR CAD	FREQUENCY	PERCENTAGE
YES	11	22%
NO	39	78%

DIAGRAM 6:

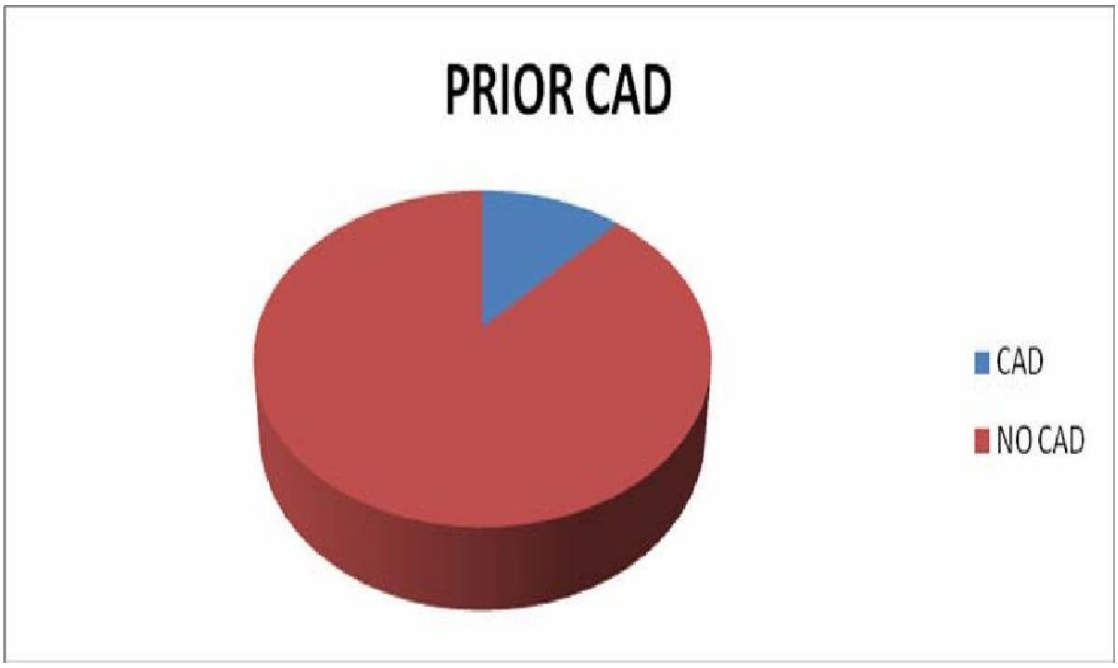


TABLE 6.2: PRIOR CAD AND MPV

MPV	Sig P value
Between groups	0.054 (SIG)

SMOKING AND ACS

TABLE 7.1:

SMOKING	FREQUENCY	PERCENTAGE
YES	28	56%
NO	22	44%

DIAGRAM 7:

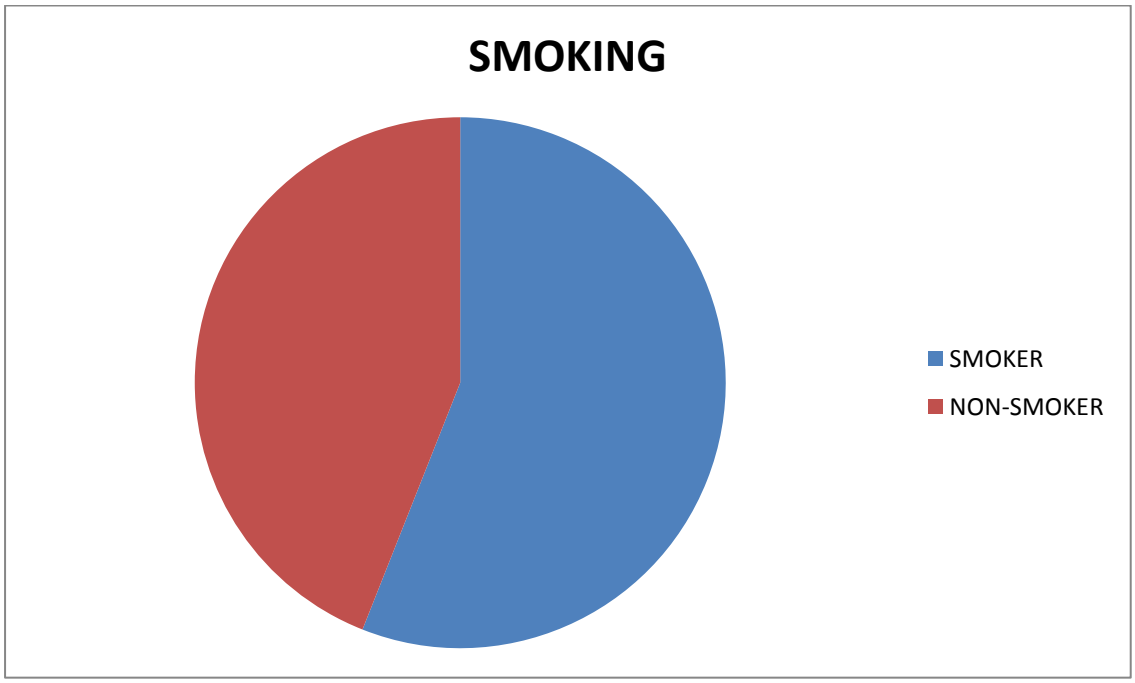


TABLE 7.2: SMOKING AND MPV

MPV	Sig P value
Between groups	0.701 (NOT SIG)

ALCOHOL AND ACS

TABLE 8.1:

ALCOHOL	FREQUENCY	PERCENTAGE
YES	37	74%
NO	13	26%

DIAGRAM 8:

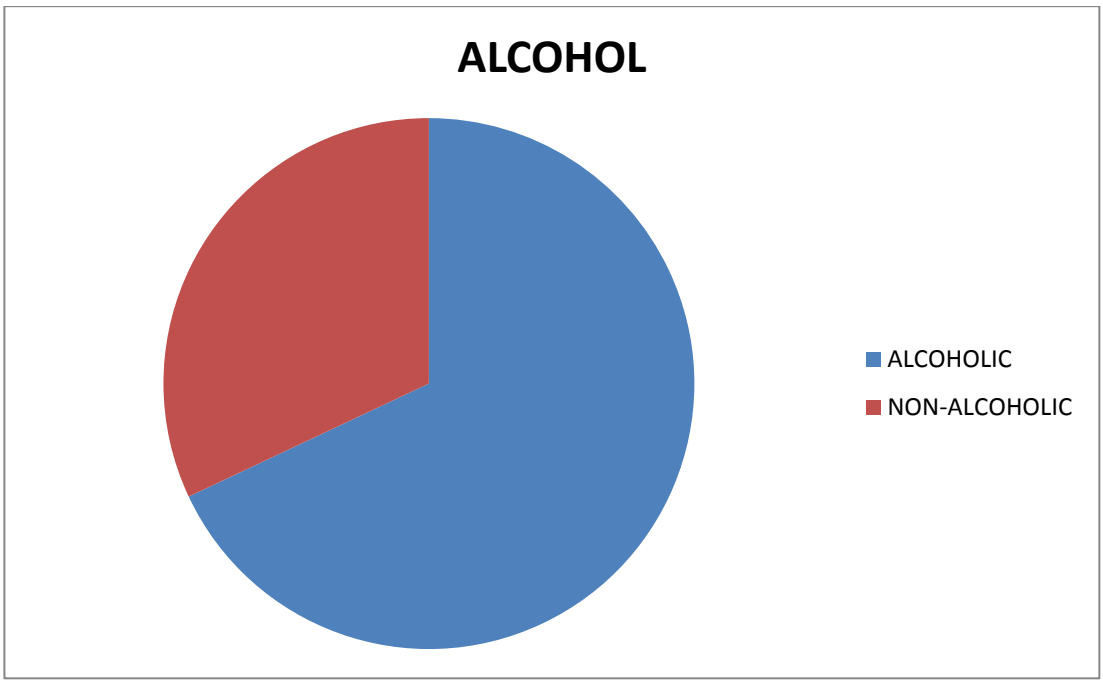


TABLE 8.2: ALCOHOL AND MPV

MPV	Sig P value
Between Groups	0.765 (NOT SIG)

KILLIP CLASSIFICATION

TABLE 9.1:

Killip Class	Frequency	Percentage	Mortality	Percentage
1	10	40%	0	0%
2	9	36%	2	67%
3	4	16%	1	33%
4	2	8%	0	0%
Total	**25**	100%	**3**	100%

DIAGRAM 9:

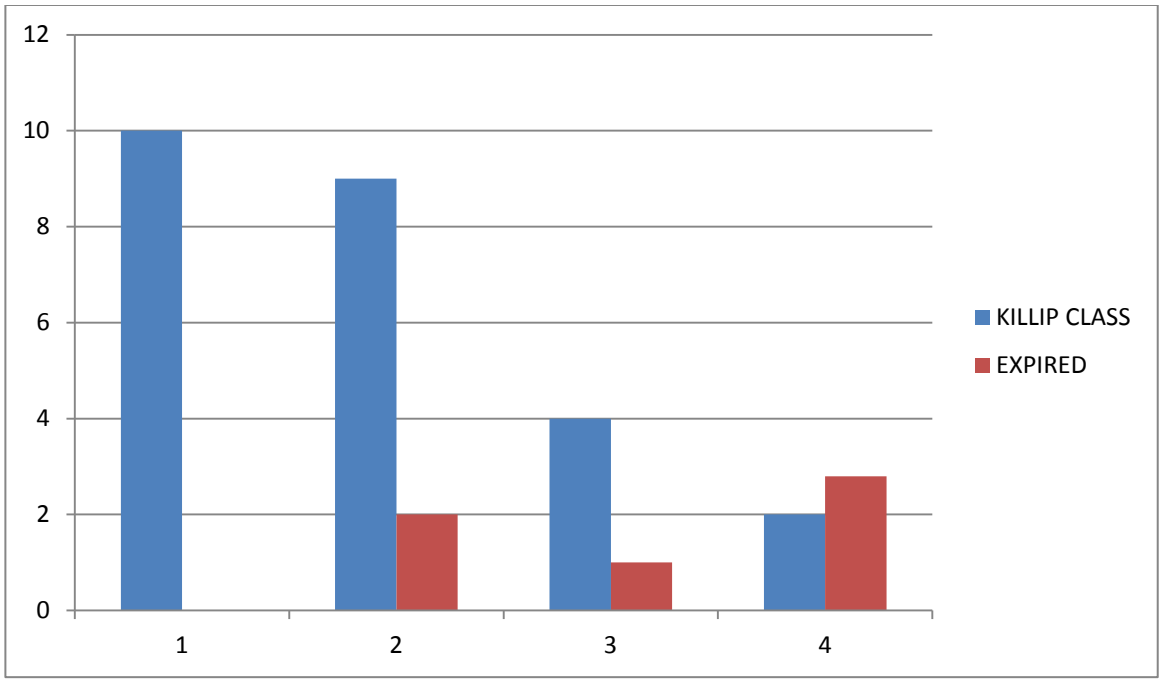


TABLE 9.2: KILLIP CLASSIFICATION AND MPV

MPV	Sig P value
Between groups	0.252 (NOT SIG)

MEAN PLATELET VOLUME IN COMPARISON

TABLE 10.1:

GROUP	**MEAN PLATELET VOLUME** (Femolitre)
MI	9.8
UNSTABLE ANGINA	9.5
STABLE ANGINA	8.4
HEALTHY CONTROL	8.2

DIAGRAM 10:

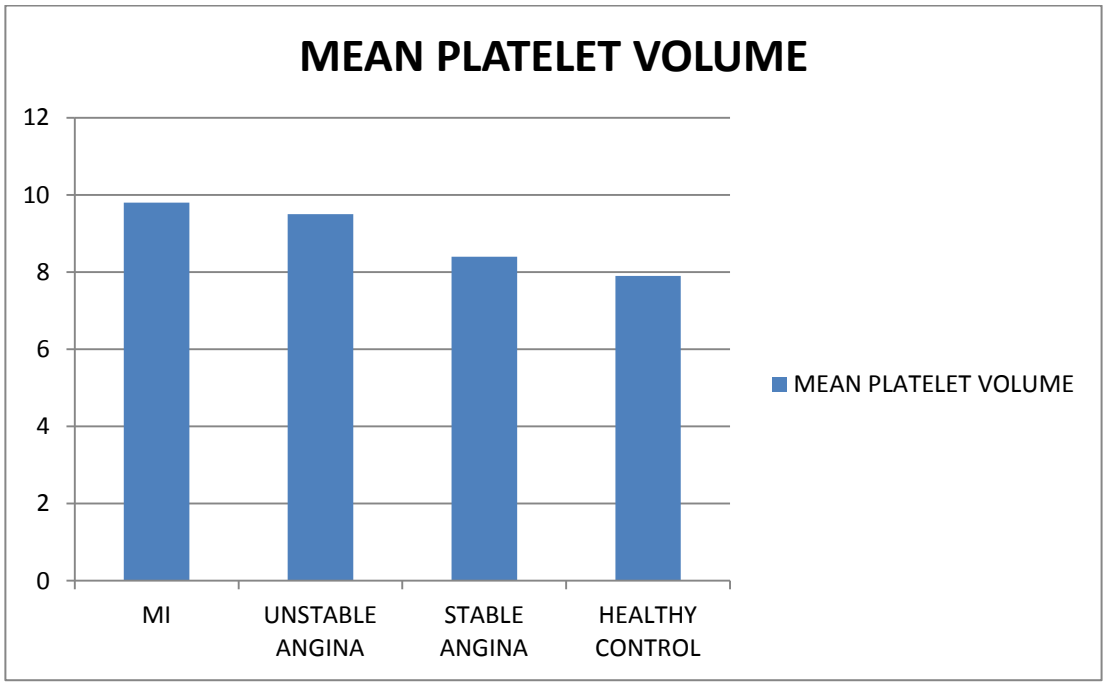


TABLE 10.2: MPV WITHIN GROUPS

ANOVA TEST

MPV	Sig P value
Between groups	<0.001

ECHOCARDIOGRAPHY FINDINGS IN ACS

TABLE 11.1:

Echo	Frequency	Percentage
NO RWMA	13	26%
RWMA	10	20%
MILD LV DYSFUNCTION	9	18%
MOD LV DYSFUNCTION	11	22%
SEVERE LV DYSFUNCTION	7	14%
TOTAL	50	100%

RWMA- REGIONAL WALL MOTION ABNORMALITY

DIAGRAM 11:

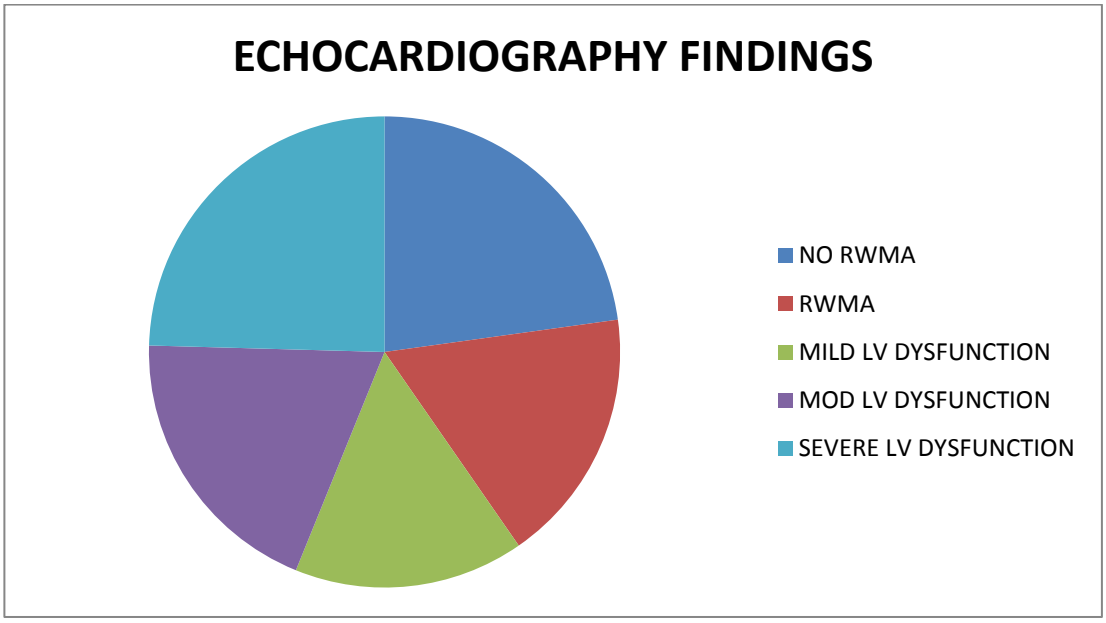


TABLE 11.2: ECHO AND MPV

MPV	Sig
Between groups	.816 (NOT SIG)

MEAN PLATELET VOLUME IN ACS

TABLE 12.1:

GROUP	MEAN PLATELET VOLUME(FEMTOLITRE)
AWMI	10.0
ASMI	9.8
IWMI	9.0
IW+PWMI	10.2
UA	9.4

DIAGRAM 12:

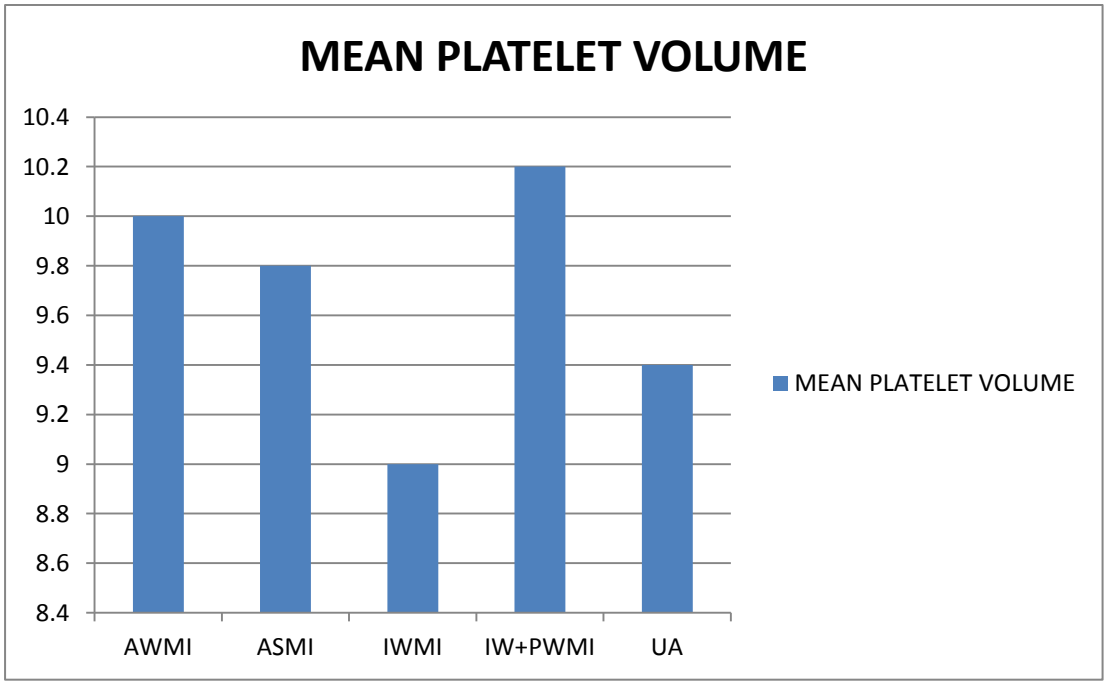


TABLE 12.2:

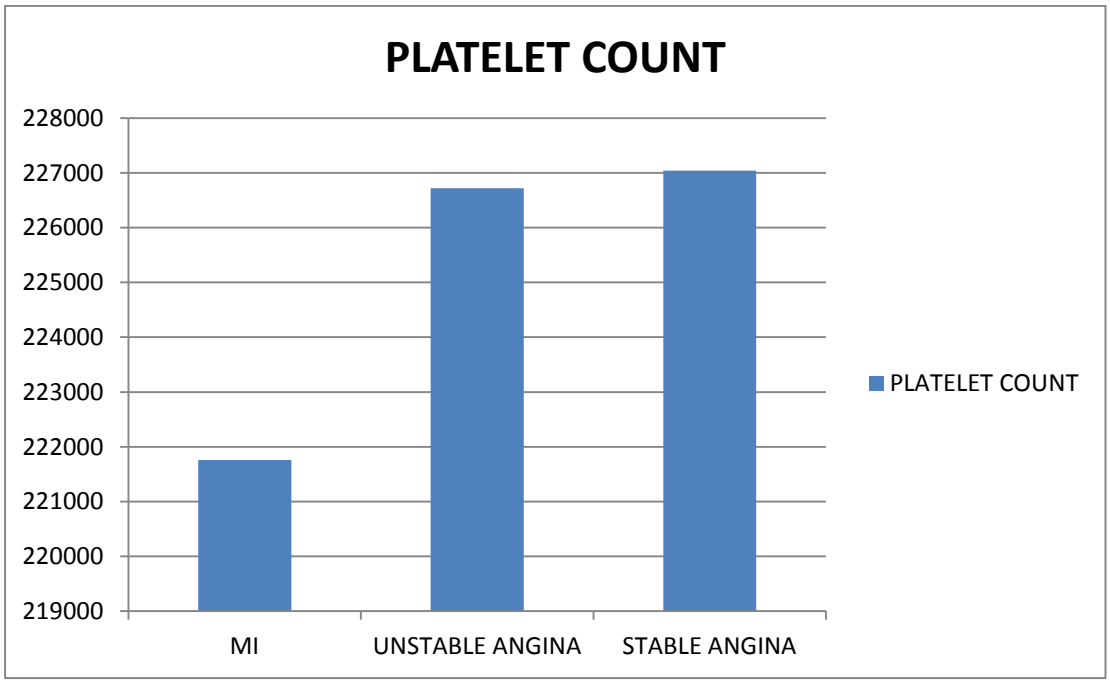
MPV	Sum of squares	Df	Mean square	F	Sig
Between groups	5.327	4	1.332	1.906	0.126 (NOT SIG)

PLATELET COUNT IN COMPARISON

TABLE 13:

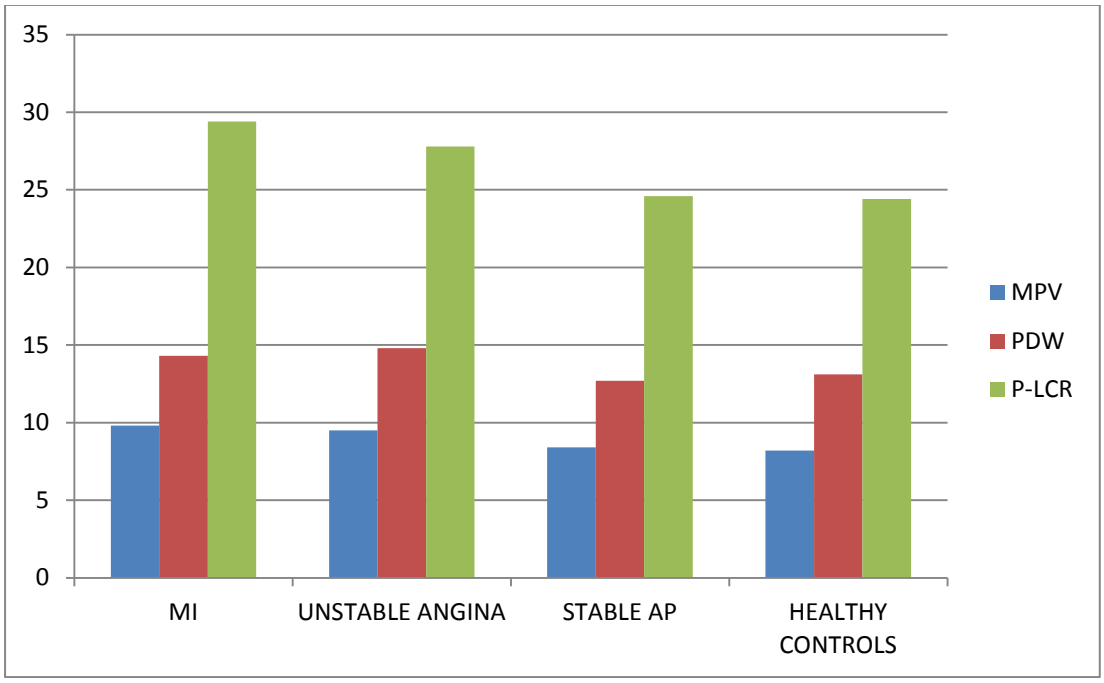
PLATELET COUNT	MEAN cells/dl
MI	2,10,840
UNSTABLE ANGINA	2,21,760
STABLE ANGINA PECTORIS	2,26,720
HEALTHY CONTROLS	2,27,040
TOTAL	2,21,590

DIAGRAM 13:



MPV PDW P-LCR IN COMPARISON

DIAGRAM 14:



MPV PDW P-LCR IN COMPARISON

TABLE 14.1:

GROUP	MPV(fl)	PDW%	P-LCR
MI	9.8	14.3	29.4
UNSTABLE ANGINA	9.5	14.8	27.8
STABLE AP	8.4	12.7	24.6
HEALTHY CONTROLS	8.2	13.1	24.4

TABLE 14.2: PLATELET VOLUME INDICES WITHIN GROUPS

ANOVA TEST

PLATELET VOLUME INDICES	SIG P VALUE
MPV Between groups	<0.001 (HIGHLY SIG)
PDW Between groups	<0.001 (HIGHLY SIG)
P-LCR Between groups	<0.001 (HIGHLY SIG)

COMPARISON OF MPV PDW P-LCR BETWEEN CASES AND CONTROLS

TABLE 15.1:

	MEAN VALUE		
	MPV(fl)	**PDW%**	**P-LCR**
CASES (MI GROUP AND UNSTABLE ANGINA GROUP)	9.6	14.5	28.6
CONTROL (STABLE ANGINA AND HEALTHY GROUP)	8.3	12.9	24.5

TABLE 15.2: COMPARISON OF MPV PDW P-LCR BETWEEN CASES AND CONTROLS

t TEST

PLATELET VOLUME INDICES	SIG P VALUE
MPV Between groups	<0.001 (HIGHLY SIG)
PDW Between groups	<0.001 (HIGHLY SIG)
P-LCR Between groups	<0.001 (HIGHLY SIG)

OUTCOME AND DEATH

TABLE 16.1:

OUTCOME	FREQUENCY	PERCENTAGE
IMPROVED	45	90%
EXPIRED	5	10%

DIAGRAM 16:

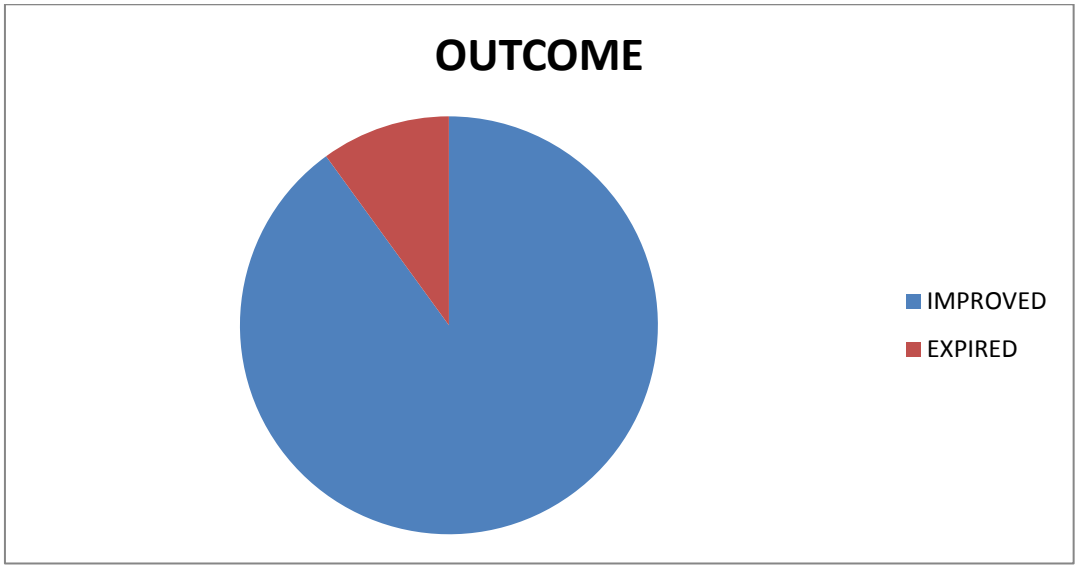


TABLE 16.2:

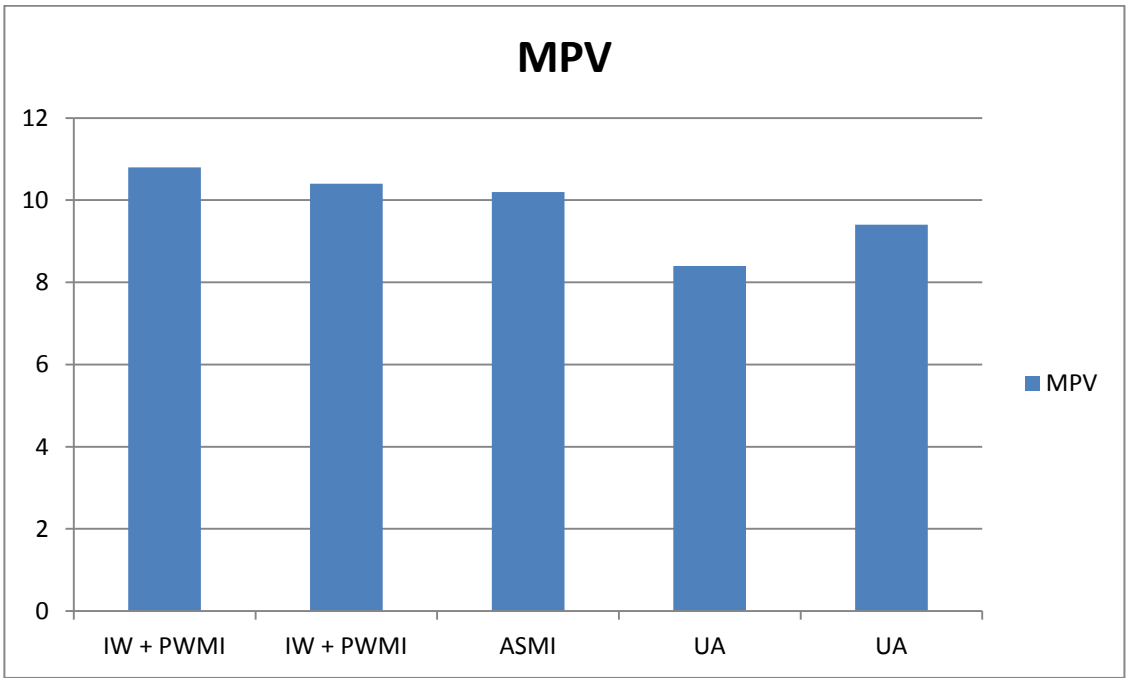
MPV	Sig P value
Between groups	0.606(NOT SIG)

MPV AND DEATH

TABLE 17:

PATIENT NAME	DEATH	MPV(femtolitre)
DEVADASS	IW+PWMI	10.8
JEYA	IW + PWMI	10.4
KUMAR	ASMI	10.2
ASHOK	UA	8.4
GUNASEKARAN	UA	9.4

DIAGRAM 17:



COMPARISON OF GROUPS BY MPV

TABLE 18:

			P VALUE	SIG/NOT SIG
MPV	MI GROUP 9.8 ± 0.9	UNSTABLE GROUP 9.5 ± 0.8	0.284	NOT SIG
	UNSTABLE GROUP 9.5 ± 0.8	STABLE GROUP 8.4 ± 0.6	<0.001	HIGHLY SIG
	STABLE GROUP 8.4 ± 0.6	HEALTHY GROUP 8.2± 0.6	0.817	NOT SIG
	HEALTHY GROUP 8.2± 0.6	MI GROUP 9.8 ± 0.9	<0.001	HIGHLY SIG
	UNSTABLE GROUP 9.5 ± 0.8	HEALTHY GROUP 8.2± 0.6	<0.001	HIGHLY SIG
	MI GROUP 9.8 ± 0.9	STABLE GROUP 8.4 ± 0.6	<0.001	HIGHLY SIG

SIG- SIGNIFICANT

COMPARISON OF GROUPS BY PDW

TABLE 19:

			P VALUE	SIG/NOT SIG
PDW	MI GROUP 14.3 ± 0.2	UNSTABLE GROUP 14.8 ± 0.7	0.752	NOT SIG
	UNSTABLE GROUP 14.8 ± 0.7	STABLE GROUP 12.7 ± 0.2	0.001	SIG
	STABLE GROUP 12.7 ± 0.2	HEALTHY GROUP 13.1 ± 0.2	0.877	NOT SIG
	HEALTHY GROUP 13.1 ± 0.2	MI GROUP 14.3 ± 0.2	0.133	NOT SIG
	UNSTABLE GROUP 14.8 ± 0.7	HEALTHY GROUP 13.1 ± 0.2	0.010	HIGHLY SIG
	MI GROUP 14.3 ± 0.2	STABLE GROUP 12.7 ± 0.2	0.021	SIG

SIG- SIGNIFICANT

COMPARISON OF GROUPS BY P-LCR

TABLE 20:

			P VALUE	SIG/NOT SIG
P-LCR	MI GROUP 29.4 ± 1.0	UNSTABLE GROUP 27.8 ± 0.6	0.258	NOT SIG
	UNSTABLE GROUP 27.8 ± 0.6	STABLE GROUP 24.6 ± 0.6	0.002	SIG
	STABLE GROUP 24.6 ± 0.6	HEALTHY GROUP 23.4± 0.3	0.992	NOT SIG
	HEALTHY GROUP 23.4± 0.3	MI GROUP 29.4 ± 1.0	<0.001	HIGHLY SIG
	UNSTABLE GROUP 27.8 ± 0.6	HEALTHY GROUP 23.4± 0.3	0.001	HIGHLY SIG
	MI GROUP 27.8 ± 0.6	STABLE GROUP 24.6 ± 0.6	<0.001	HIGHLY SIG

SIG- SIGNIFICANT

DISCUSSION

In our study, four groups were taken into account such as MI group, unstable angina, stable angina pectoris and healthy controls. Cases (acute coronary syndrome) include MI group and unstable angina. Controls include stable angina pectoris and healthy controls.

In our study the significance of platelet volume indices in cases (acute coronary syndrome) is the prime concern and they were compared with controls (stable angina pectoris and healthy controls).

Blood samples for platelet volume indices were taken at the time of admission in cases. Platelet volume indices was analysed by automatic analyzer. Statistical significance in Platelet volume indices was analysed between cases and controls.

AGE AND MPV IN ACS

Mean age of patients who sustained ACUTE CORONARY SYNDROME was 45 years in my study. No statistical significance was noted in MPV between age groups in relation to cases (p value is .765 i.e. >.05).

SEX DISTRIBUTION AND MPV IN ACS:

Out of 50 patients 40 (80%) were males and 10 (20%) were female. No statistical significant significance was detected in MPV between the two sexes in relation to ACS (p value is .206 i.e. >.05).

TYPE OF ACS

In my study, out of 50 ACS patients, 12 (25%) had AWMI, 5 (10%) had ASMI, 4(8%) had IW+PWMI and 25 (50%) had UNSTABLE ANGINA.

HYPERTENSION AND MPV IN ACS

31(62%) were hypertensive and 19(38%) were non-hypertensive. No statistical significance was detected in MPV between the hypertensive and non-hypertensive in relation to ACS (p value is 0.346 i.e. >.05).

DIABETES AND MPV IN ACS

34 (68%) were diabetic and 16(32%) were non diabetic. No statistical significance was detected in MPV between diabetics and non-diabetics in relation to ACS (p value is .959 i.e. >.05).

SMOKING AND MPV IN ACS

28(56%) were smoker out of 40 males. None of the females were smokers. 22(44%) were non-smoker. No statistical significance was noted in MPV between smokers and non-smokers in relation to ACS (p value is .106 i.e. >.05)

ALCOHOL AND MPV IN ACS

37 (74%) were consuming alcohol and 13(26%) did not consume alcohol. No statistical significance was detected in MPV between alcoholics and non-alcoholics in relation to ACS (p value is .765 i.e. >.05).

KILLIP CLASSIFICATION AND MPV IN ACUTE MYOCARDIAL INFARCTION

Patients were divided into 4 based on this classification. 10 (40%) were in Class 1, 9 (36%) were in class 2, 4 (16%) were in class 3 and 2(8%) were in class 4. No statistical significance was detected in MPV between the killip groups in relation to ACS (p value is .252 i.e. >.05).

ECHO FINDINGS AND MPV IN ACS

In our study, echo findings were analysed. In this, 13 (26%) had NO RWMA, 10(20%) had RWMA, 9(18%) had MILD LV

DYSFUNCTION, 11(22%) had MODERATE LV DYSFUNCTION, 7(14%) had SEVERE LV DYSFUNCTION. No statistical significance was detected in MPV between echo findings in relation to ACS (p value is .816 i.e. >.05).

MEAN PLATELET VOLUME

Our study showed that mean platelet volume (hectolitres) in MI group was 9.8, UNSTABLE ANGINA group was 9.5, STABLE AP group was 8.4 and HEALTHY CONTROLS was 8.2. Mean platelet volume were comparable between four groups with high statistically difference of 0.000 (<0.5).

Mean platelet volume were comparable between Cases (acute coronary syndrome) includes MI group and unstable angina and Controls includes stable angina pectoris and healthy controls with high statistically difference of 0.000 (<0.5).

In a similar study[4] which compared AMI and unstable AP, MPV in unstable AP was 9.0 ± 1.0 fl and 8.9 ± 0.8 fl in acute myocardial infarction. No statistical significance was detected in MPV between these (difference p=0.999) two groups.

In control group was 7.2 ± 0.6 fl and in stable AP patients, MPV was 7.5 ± 0.6 fl, respectively. No statistical significance was detected in MPV between these two groups (difference p=0.126).

When stable angina pectoris and control groups were compared to each of AMI cases and unstable angina, it was found that MPV was increased in AMI cases and unstable angina.

Here in our study too, there is statistically significant difference between cases (MI group and UNSTABLE ANGINA) and other two groups which is similar to the above cited study[4].

In the study by Ender et al[10], it is concluded that MPV was found to be increased who those with AMI patients on comparison with stable AP patients and this result was consistent with our study too.

In the study comparing AMI patients to control groups and stable angina pectoris conducted by Kishk et al[24] revealed that lower platelet count and higher MPV to be associated with AMI group than the stable angina pectoris and control groups. In our study, mean platelet volume was higher in AMI group and platelet count was also reduced than others.

In another study conducted by Puzzili et al[36] , concluded that MPV was higher side in unstable angina pectoris patients, compared to control groups and stable angina which is consistent with our study.

MEAN PLATELET VOLUME IN ACS

In our study mean platelet volume in AWMI was 9.6, ASMI was 9.6, IWMI was 9.7, IW+PWMI was 10.0, IW+PW+RVMI was 9.8 and UA was 9.5. No statistical significance was detected in MPV between the various types of ACS (p value is .889 i.e. >.05).

PLATELET COUNT AND ACS

In our study, platelet counts in four groups were analysed. Mean value in AMI group was 2,10,840 cells/dl, UA group was 2.21,760 STABLE AP was 2,26,720 cells/dl and HEALTHY CONTROLS was 2,27,040 cells/dl. While comparing between the groups there was no statistical difference between any groups. But in our study we compared mean platelet count in AMI patients to stable AP and control groups and we detected that the AMI group had lower platelet count comparing with stable AP and control group.

Hence in our study the results were convincing while comparing to the Kishk et al[24] in terms of platelet count.

PLATELET DISTRIBUTION WIDTH AND ACS

In our study mean value of in cases (AMI group and UNSTABLE ANGINA group) was 14.5% and controls (STABLE ANGINA and Healthy group) were 12.9%. Statistically high significance was detected in PDW between cases and control.(p value - <0.001).

PLATELET-LARGE CELL RATIO AND ACS

In our study mean value of P-LCR in cases (AMI group and UNSTABLEANGINA group) was 28.6 and controls (STABLE ANGINA and Healthy group) were 24.5. Statistically high significance was detected in PDW between cases and control.(p value - <0.001).

OUTCOME AND MPV IN ACS

In our study, 5(10%) patients expired and 45(90%) survived out of 50 in the ACS group. No Statistical significance was detected in MPV between the outcomes in relation to ACS (p value is .606 i.e. < .05).

CONCLUSION

It is revealed from the study that Platelet volume indices is increased in cases (AMI group and UNSTABLEANGINA group) in comparison with controls (STABLE ANGINA and Healthy group). Statistical difference has been showed in PVI between cases and controls.

LIMITATIONS OF STUDY

1) PVI has been found to be dependent on a number of variables like

   a. The time taken for analysis after venipuncture,

   b. The method of analysis,

   c. The Anticoagulant used

   d. Temperature of Specimen storage

2) When compared to other studies , the population studied is small.

3) Cardiac biomarkers were not quantified for all the patients.

4) Coronary angiography was not carried out in all the patients.

CONCLUSION

1) Mean platelet volume was found to be increased in ACS group
1) When compared to Stable angina pectoris and Healthy controls Platelet volume indices is found to be increased in patients with ACS (AMI group and UNSTABLE ANGINA group).
2) Statistically significant difference in platelet volume indices was found to exist between cases (AMI group and UNSTABLE ANGINA group) and controls (Stable angina pectoris and Healthy controls).
3) PVI is a feasible and easy reliable test , thus it can be used for the initial evaluation of patients admitted with ACS along with other cardiac biomarkers.

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ANNEXURES

PROFORMA

NAME:

AGE:

SEX: PHONE NO:

OCCUPATION:

ADDRESS:

EDUCATIONAL STATUS OF THE PATIENT:

CHIEF COMPLAINTS:

HISTORY OF PRESENTING ILLNESS:

DURATION OF CHEST PAIN:

PAST HISTROY: DM/HT/PRIOR CAD

PERSONAL HISTORY: ALCOHOLCONSUMPTION/ SMOKER

FAMILY HISTORY:

VITAL SIGN: PULSE

BP

EXAMINATION OF CVS:

EXAMINATION OF RS:

EXAMINATION OF ABDOMEN:

EXAMINATION OF CNS:

DIAGNOSIS:

KILLIP CLASS:

TREATMENT :THROMBOLYSED/HEPARINED

INVESTIGATION:

1. HEMOGLOBIN
2. TOTAL COUNT(WBC)
3. DIFFERENTIAL COUNT
4. PLATELET COUNT
5. RENAL FUNCTION TEST
6. LIVER FUNCTION TEST
7. LIPID PROFILE
8. MPV(MEAN PLATELET VOLUME)
9. PDW(PLATELET DISTRIBUTION WIDTH)
10. P-LCR(PLATELET LARGE CELL RATIO)
11. ECG
12. ECHOCARDIOGRAPHY

DURATION OF HOSPITAL STAY:

OUTCOME:

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Protocol ID No.11/01/2015 Dt. .01.2015
CERTIFICATE OF APPROVAL

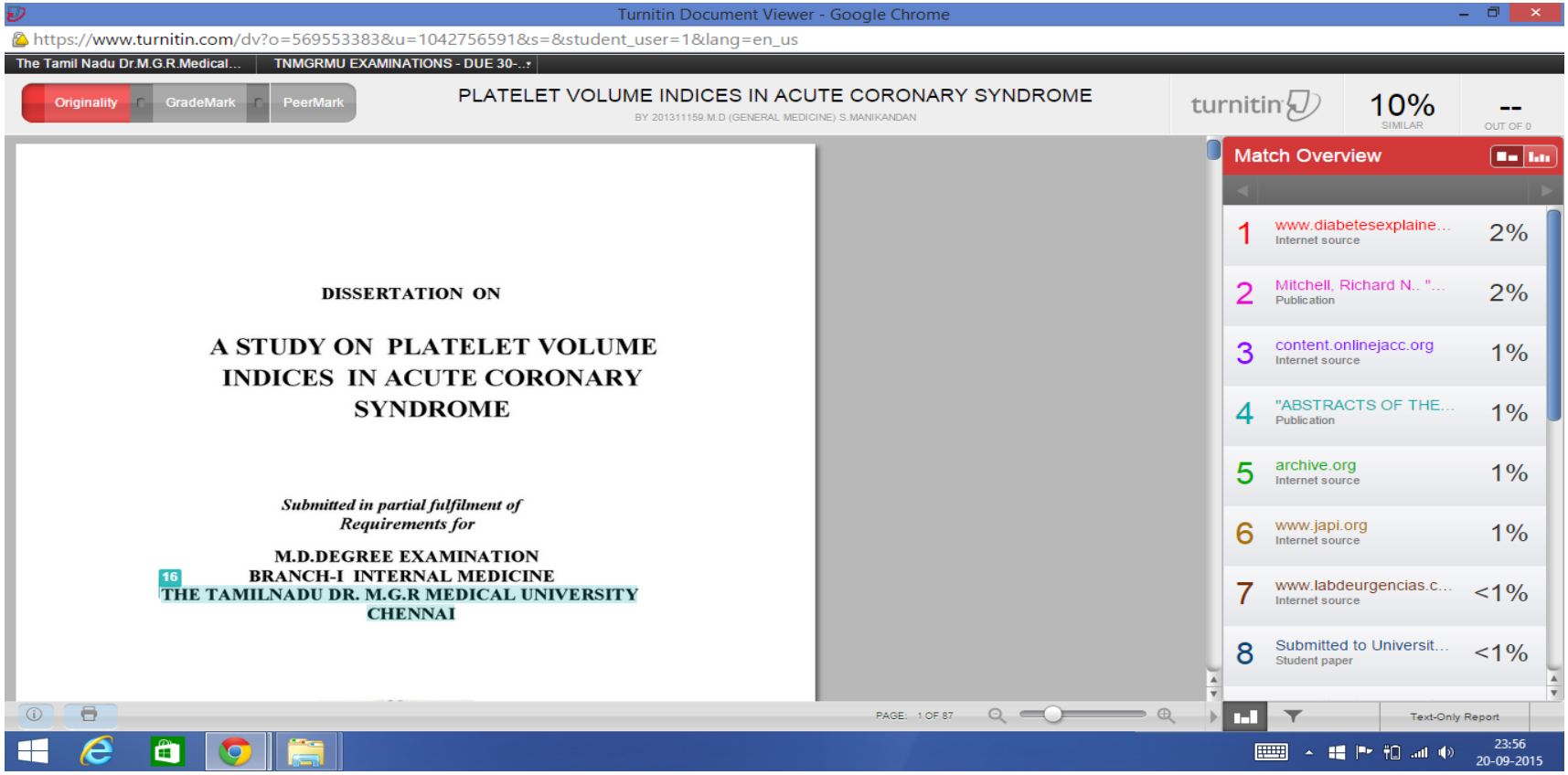
The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "Platelet volume indices in patients with acute coronary syndrome n Govt. Royapettah Hospital". -For Project Work-submitted by Dr.S.Manikandan, PG in General Medicine, KMC, Chennai- 10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

GOVT. KILPAUK MEDICAL COLLEGE
JAN 2015
Kilpauk, Chennai-10.

CHAIRMAN,
Ethical Committee
Govt.Kilpauk Medical College,Chennai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Submitted in partial fulfilment of Requirements for
M.D.DEGREE EXAMINATION
BRANCH-I INTERNAL MEDICINE
THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY
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	NAME	AGE	SEX	PRESENTATION	DURATION(days)	HYPERTENSIO	DIABETES	FHOHCAD	SMOKING	ALCOHOL	BP mmHg	PULSE	HBALC	THROMBOLYS	HEPARIN	TOTALCOLN	H	L	E	PLATELET	GLUCOSE	UREA	CREATININE	TB	DB	TC	TGL	HDL	HBP	PCW	P-LCOR	ECHO
2	GANESAN	46	M	AWMI		NO	NO																									
3	MOHAN	45	M	ASMI																												
4	EDWARD	49	F	AWMI		NO	NO																									
5	DEVADASS	38	M	NWMI		NO	NO																									
6	RAMESH	69	M	AWMI		YES	NO																									
7	SAROJA	54	F	ASMI		NO	NO																									
8	SAMUVEL	36	M	AWMI		NO	NO																									
9	MUTHUKUMAR	52	M	AWMI		NO	NO																									
10	JEYA	56	M	AWMI		NO	NO																									
11	MURALI	49	M	AWMI		NO	NO																									
12	NAGAMMAL	49	F	AWMI		NO	NO																									
13	JOTA	54	F	NWMI		NO	NO																									
14	MURUGESAN	58	M	AWMI		NO	NO																									
15	NATARAJAN	49	M	AWMI		NO	NO																									
16	KUMAR	58	M	ASMI		NO	NO																									
17	VARADARAJAN	49	M	AWMI		NO	NO																									
18	JOHNSON	49	M	AWMI		NO	NO																									
19	RAJAJI	56	M	NWMI		NO	NO																									
20	SIVAKAMI	47	F	ASMI		NO	NO																									
21	CHANDRASEKAR	38	M	AWMI		NO	NO																									
22	KRISHNAMOORTHY	42	M	NWMI		NO	NO																									
23	MARY	49	F	AWMI		NO	NO																									
24	RAJENDRAN	44	M	AWMI		NO	NO																									
25	JOTI	31	F	ASMI		NO	NO																									
26	JAYAKUMAR	49	M	AWMI		NO	NO																									
27	DEVAR	31	M	AWMI		NO	NO																									
28	THIRUMALAI	49	M	AWMI		NO	NO																									
29	RAMACHANDRAN	58	M	AWMI		NO	NO																									
30	PERUMAL	32	M	AWMI		NO	NO																									
31	CHANDRASEKAR	36	M	AWMI		NO	NO																									
32	GOPALAKRISHNAN	49	M	AWMI		NO	NO																									
33	ANNAMALAI	67	F	AWMI		NO	NO																									
34	ASHOK	58	M	AWMI		NO	NO																									
35	DEVARAJ	58	M	AWMI		NO	NO																									
36	KAMARAJ	49	M	AWMI		NO	NO																									
37	ANNAMALAI	49	M	AWMI		NO	NO																									
38	DURAI	49	M	AWMI		NO	NO																									
39	PERUMAL	58	M	AWMI		NO	NO																									
40	GUNASEKARAN	49	M	AWMI		NO	NO																									
41	MAHENDRAN	49	M	AWMI		NO	NO																									
42	ANBU	49	M	AWMI		NO	NO																									
43	PRAKASH	56	M	AWMI		NO	NO																									
44	PUSHPARAJ	49	M	AWMI		NO	NO																									
45	VASUDEVAN	54	M	AWMI		NO	NO																									
46	RAJASIRAMANI	54	M	AWMI		NO	NO																									
47	JAYARAJ	49	M	AWMI		NO	NO																									
48	ANBAL	49	F	ASMI		NO	NO																									
49	VATH	53	M	AWMI		NO	NO																									
50	THANIGAVEL	49	M	AWMI		NO	NO																									
51	PARVATHY	49	F	AWMI		NO	NO																									

32	SHEIKMOHAMED	40	M	SAP		YES	YES	YES	HO	11+00	50			7400	24	30	5	12000+	96	30	0.0	0.0	0.1	140	120	64	7.4	11.0	23.2
33	MUTHARAL	35	M	SAP		NO	NO	NO	NO	10+00	50			5400	24	24	5	9+000+	102	30	0.0	0.0	0.2	154	140	66	6.8	10.4	23.0
34	MANIKANDAN	34	M	SAP		NO	YES	YES	YES	14+00	100			7400	24	30	5	9+000+	110	30	0.0	0.0	0.1	140	110	54	7.4	11.0	23.2
35	ASHOK	45	M	SAP		YES	YES	NO	YES	10+00	50			5400	22	26	5	9+000+	90	30	1	1	0.1	140	100	60	*	10.6	23.2
36	JAYALAKSHMI	34	F	SAP		YES	NO	NO	NO	10+00	50			5400	24	26	4	10400+	90	30	0.7	0.7	0.1	140	140	44	6.8	10.0	23.2
37	ARUN	34	M	SAP		YES	NO	YES	YES	11+00	64			7200	24	30	5	8220+	100	30	0.0	0.0	0.2	154	100	40	6.6	10.0	23.0
38	KADHAR	34	M	SAP		NO	NO	NO	YES	11+00	50			7400	24	30	4	12000+	120	30	0.0	0.0	0.2	170	110	50	7.4	10.2	23.0
39	SHANTI	34	F	SAP		NO	YES	NO	NO	10+00	50			5200	24	30	4	10000+	100	30	0.5	0.1	0.1	140	100	40	6.6	10.4	23.2
40	LAKSHMI	42	F	SAP		YES	YES	NO	NO	10+00+	50			5400	24	30	4	9+000+	104	30	0.4	0.0	0.1	140	140	54	6.7	11.0	24.0
41	VISVANATHAN	40	M	SAP		YES	NO	NO	NO	14+00	64			7400	24	30	5	9+000+	99	30	1	0.0	0.1	140	100	44	6.8	11	23.2
42	MUTHUMADAN	34	M	SAP		YES	NO	YES	NO	10+00	100			5400	27	30	5	9+000+	140	30	0.0	1	0.1	140	104	40	7.4	11.0	23.2
43	MARIAPPAN	35	M	SAP		NO	YES	YES	NO	10+00	50			7400	24	24	5	10000+	110	30	0.0	0.0	0.1	154	110	60	6.8	10.6	24.0
44	SENTHILNAT	34	M	SAP		YES	NO	NO	YES	10+00	64			5400	24	30	6	10000+	90	30	0.0	0.0	0.1	140	140	40	6.4	10.0	23.2
45	THULASI	45	F	SAP		YES	NO	NO	NO	10+00	50			7400	24	30	4	8400+	105	30	0.7	0.7	0.1	170	100	40	7.4	11.0	23.2
46	DAKSHAN	40	M	SAP		NO	YES	NO	YES	10+00	50			5400	24	30	1	8500+	240	30	0.0	1	0.2	140	120	40	6.4	10.6	24.0
47	ACHAMMAL	32	M	SAP		YES	YES	YES	YES	10+00	50			7400	27	30	5	10000+	90	30	0.0	1.1	0.3	140	100	50	7.1	10.0	23.2
48	JAYARAMAN	34	M	SAP		YES	NO	YES	YES	10+00	50			7200	22	26	5	10000+	104	30	0.0	0.0	0.2	140	140	50	6.4	10.2	23.4
49	KAVITHA	34	F	SAP		NO	NO	NO	NO	10+00	50			5400	21	26	5	8000+	90	30	1	0.1	0.1	140	100	60	7.1	11.0	23.0
50	DHANUSH SELVAM	23	M	SAP		NO	YES	NO	YES	10+00	50			6100	24	30	5	8500+	145	30	0.0	0.0	0.1	140	100	60	7.4	10	23.2
51	DADU	34	M	SAP		YES	NO	YES	YES	14+00	64			5400	24	30	5	10000+	84	30	0.0	0.0	0.2	140	120	50	6.8	10.6	23.0
52	KANNAN	45	M	SAP		NO	NO	NO	NO	11+00	50			5200	25	30	5	12000+	90	30	0.0	1	0.1	140	100	67	6.4	10.4	23.2
53	UMAPATHI	31	M	SAP		NO	NO	NO	NO	10+00	50			7400	24	24	6	10000+	90	30	1	0.1	0.1	154	110	46	6.8	11.2	24.1
54	KAMALA	48	F	SAP		YES	NO	NO	NO	10+00	50			7400	24	30	5	9+000+	110	30	0.0	1.1	0.2	170	100	60	7.4	10.8	23.4
55	SRINIVASAN	38	M	SAP		YES	NO	YES	YES	14+00	50			7600	24	30	5	9+000+	95	30	0.7	0.7	0.1	240	150	60	6.8	11	23.1
56	SURESHRAJU	41	M	SAP		YES	NO	NO	NO	14+00	50			7400	22	26	5	10000+	90	30	0.0	0.0	0.2	170	120	60	7.0	10.4	23.4
57	ARUMUGAM	41	M	HP		YES	YES	NO	NO	11+00	50			7400	21	30	4	11000+	110	30	0.0	1.1	0.2	220	140	64	*	11.2	23.4
58	YESUDOSS	31	M	HP		YES	NO	NO	YES	14+00	64			9400	27	30	5	13000+	135	30	1	0.1	0.1	170	80	60	6.4	10.6	23.2
59	RUKMANI	41	F	HP		YES	YES	NO	NO	14+00	50			7400	24	22	4	10400+	220	30	0.7	0.0	0.2	140	140	40	7.0	10.8	23.6
60	DINESH KUMAR	44	M	HP		YES	YES	YES	YES	11+00	64			7400	22	22	6	11800+	90	30	0.7	0.0	0.2	170	120	64	*	10.2	23.6
61	SANKAR	34	M	HP		NO	NO	YES	YES	11+00	60			7600	24	26	4	10000+	102	30	0.0	1.1	0.1	140	100	64	6.2	10.6	24.0
62	ANGAMMAL	34	F	HP		NO	YES	NO	NO	10+00	50			5400	21	20	4	10000+	102	30	0.0	1	0.2	154	120	40	6.8	10.4	23.4
63	CHINNASAMY	48	M	HP		YES	YES	YES	YES	10+00	64			5400	21	20	5	10400+	145	30	0.0	0.0	0.1	140	100	44	6.2	10.4	23.6
64	GOPALAKRISHNA	41	M	HP		YES	YES	NO	NO	10+00	50			7400	21	20	5	10000+	145	30	0.0	0.7	0.1	140	120	46	7.4	10.2	23.6
65	SANTHAKUMARI	31	F	HP		NO	YES	NO	NO	10+00	50			5400	24	20	5	10000+	96	30	0.7	0.7	0.1	154	120	44	7.2	11.0	23.4
66	NAGANATHAN	48	M	HP		YES	YES	NO	NO	14+00	67			7600	24	20	5	10400+	105	30	0.0	0.0	0.2	140	120	50	6.2	11.2	23.8
67	BHARANI	41	F	HP		NO	YES	NO	NO	11+00	72			9100	24	20	5	10000+	120	30	0.0	1	0.1	170	110	50	6.4	10.0	23.6
68	CHANDRAN	42	M	HP		YES	YES	YES	YES	10+00	64			7400	24	22	4	10400+	100	30	0.0	0.0	0.1	240	160	46	6.4	10.2	23.2
69	SELVARAJ	34	M	HP		YES	YES	YES	YES	11+00	60			7400	24	22	4	10000+	94	30	0.0	0.0	0.1	154	140	44	7.2	10.4	23.4
70	BALAKRISHNAN	41	M	HP		NO	YES	NO	YES	11+00	64			7400	21	22	4	10000+	90	30	0.7	0.7	0.1	240	100	64	*	10.2	24.0
71	SHANMUGAM	37	M	HP		YES	NO	NO	NO	10+00	50			9400	21	20	5	10400+	90	30	0.0	1	0.2	140	170	40	7.0	10.4	23.4
72	VIMALA	34	F	HP		YES	NO	NO	NO	10+00	50			7400	24	20	5	10000+	140	30	0.0	0.0	0.1	220	120	40	7.0	10.2	24
73	BASKARADEEN	34	M	HP		NO	NO	YES	NO	14+00	50			7400	21	26	6	10000+	90	30	0.0	0.0	0.2	154	100	60	6.2	10.2	24.0
74	JOSEPH	48	M	HP		YES	YES	NO	NO	11+00	50			7200	24	20	5	10400+	70	30	0.7	0.7	0.1	140	110	60	6.0	10.4	23.4
75	JAYASINGH	34	M	HP		NO	NO	YES	YES	10+00	72			9100	24	22	4	10000+	102	30	0.0	0.7	0.1	220	140	62	6.4	11.2	23.2
76	GOURI	41	F	HP		NO	NO	NO	NO	11+00	50			7400	24	20	1	10400+	94	30	1	0.0	0.2	170	110	40	7.0	11.0	23.4
77	DEVANDRAN	41	M	HP		YES	YES	YES	YES	11+00	50			7400	24	20	4	10000+	104	30	0.7	1	0.1	170	120	60	7.2	10.2	23.6
78	THIRUMURTHI	52	M	HP		YES	YES	NO	NO	10+00	67			8200	24	30	4	10000+	150	30	0.7	0.7	0.2	170	100	60	6.4	10.4	23.2
79	MOSES	52	M	HP		YES	NO	YES	YES	10+00	50			7600	24	30	4	10400+	90	30	1.1	1	0.1	240	120	60	7.2	10.8	23.6
80	CHANDRAN	34	M	HP		NO	NO	YES	YES	10+00	50			8400	24	26	4	10600+	80	30	0.8	0.8	0.1	150	100	60	6.2	10.4	23.6
81	VENKATARAMANAN	34	M	HP		YES	YES	YES	YES	10+00	50			7400	24	30	5	10400+	110	30	0.7	0.8	0.1	140	140	50	6.6	10.8	23.6